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EDITED BY

FRED. J. SMITH, M.D., M.R.C.P.,

ASSISTANT PHYSICIAN, LONDON HOSPITAL

LIST OF CONTRIBUTORS.

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A CLINICAL LECTURE

ON

INJURY OF THE BRAIN FROM CONCUSSION.

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BY

THOMAS BRYANT, M.Ch., F.R.C.S.E. and I.,

Consulting Surgeon to Guy's Hospital.

GENTLEMEN,—I propose to bring before you to-day the important subject of "injury of the brain from 'concussion,'" as I am convinced that this term "concussion of the brain," as generally employed, is as confusing to the medical as it is to the lay mind, and that, by being so, its use leads too often to a misapprehension of the nature of many of the head cases the surgeon has to treat, and consequently to their bad treatment. In support of this view you might have recently seen a case of head mischief not of traumatic origin reported in the papers as one of concussion.

To start with, let us therefore clearly understand that what we mean by "concussion of the brain," is injury to the brain as the result of some shaking force applied from without, either by way of a fall from above on to the head, or by way of a blow applied to the head.

As a preliminary reflection I would, however, wish to emphasize a fact which all surgeons should bear in mind, viz.: that the effects of a force applied to the skull are much influenced by its thickness, and that in this matter there is in cranial bones great diversity. Under these circumstances, a slight blow on a thin skull may bring about a fracture, and no general cerebral injury, since the vibrations originated by the force of impact are expended locally upon the part struck, and are not carried along the bony basal ridges so as to vibrate in the brain structure, and bring about mischief. Whereas, whilst a severe external force may fail to cause a fracture of a thick skull, it may start such intense vibrations within the cranium as to cause bruising or laceration of the brain, either of its

surface or of its substance, at a point remote from the seat of impact, and even at times produce laceration of the venous sinuses or of the middle meningeal artery. In the former case where there is a local fracture the injury on the face of it looks severe, whereas it may be comparatively trivial, since there is no cerebral injury. In the latter instance, where there is neither wound nor fracture, the injury may appear slight, although in reality it is one which bodes death from cerebral mischief.

To say that injuries of the head should always be estimated *primarily* with reference to the amount of damage the cranial contents have sustained, and *secondarily* with reference to the risk of their becoming involved, is to say what all sound surgeons of experience believe, and make the basis of their treatment. And yet students are taught to think that scalp wounds, fractures of the skull, hæmorrhage beneath the bone, concussion and compression of the brain, and inflammation of the brain, are separate and independent affections, with diagnostic symptoms which can be tabulated. The surgeon, however, who goes to the post-mortem room for information, knows too well that in every case of cranial injury of any importance, there are certain brain changes common to most, if not all, which should be fully recognised and taken into account in its diagnosis, prognosis, or treatment; and that, should either a fracture, with or without depressed bone or intracranial hæmorrhage, coexist in the same case, such a condition had better be regarded as a complication of the common factor, rather than as one which stands alone. For example, a man falls or receives a blow upon the head, and is for a time "stunned" —that is, rendered more or less senseless from paralysis of brain function; he is said to be suffering from "concussion of the brain," whatever that term may mean. Another man, as a result of the same kind of accident, receives in addition to the "stunning" a scalp wound, with a fissure either in the vertex or base of his cranium; and he is described as one who is suffering from a compound fracture of the skull, either of the vertex, base, or of both. A third man comes under observation in the same state of so-called concussion, but with a depressed fracture of bone, complicated or not

with a scalp wound, and as a result of this depression there may or may not be other symptoms, or those present may be intensified. Under either circumstance, however, his case is described as one of depressed fracture of the skull, giving rise to compression of the brain. Yet in all these different classes of cases there is one common injury, one common source of danger, present or remote—viz.: the condition of the brain which is associated with the injury, and which has been brought about by the "stunning" force. If the general cerebral injury be trivial, the local complication of a scalp wound, or even of a fissured or depressed fracture, is, although serious, comparatively unimportant; if it be of a grave nature, the local complication must, however great, sink into insignificance.

What, then, it may be asked, is the condition of a brain in a state of so-called concussion? Let us inquire. Concussion of the brain means, in a physiological sense, a sudden and more or less complete arrest of the brain's mental and physical functions, brought about by external violence. The brain in its bony case has been made to vibrate more or less roughly either by some general shaking of the whole frame, or by some violence applied directly or conveyed indirectly to the cranium as a localised or diffused force, the effects of this force upon the brain being of necessity proportional to its concentration and intensity, and in a degree to the age and healthiness of the brain structure and the thickness of the cranial wall. Thus, a concentrated blow with a blunt or edged instrument, probably, and in a thin skull certainly, spends its force in producing a local cranial or cerebral injury; whereas, any force of a diffused nature, directly or indirectly applied, and whether causing a fracture of the cranium or not, more likely brings about some structural change in the cerebral tissue, remote from, rather than at, the seat of impact. And should the brain or its vessels, in either case, have undergone senile or any morbid change, it will be more prone to suffer seriously from such external violence than a healthy organ. In every case, therefore, of injury to the head, the brain is made to vibrate more or less forcibly; when the vibrations are feeble, the injury to the brain structure resulting therefrom is but slight; when they are severe the mischief may be great. The complication of a fissure or fracture of the skull does not of itself, of necessity, tend to aggravate the cerebral mischief; although its presence may be regarded as a measure of the force which has brought it about. In all cases of cranial injury, therefore, the conclusion is clear that the cerebral mischief is the common factor, and the one important point to be taken into account.

This conclusion subsequently leads to the important question, What are the changes found in the brain after so-called concussion of its substance, or rather shaking of its structure? What are the structural changes, if any, which can be made out on the post-mortem table? For, of course, an answer to these questions can only be given from the observation of cases which have proved fatal, either directly from the injury, or at some remote period after the injury from other causes. Happily, the answer to this question is neither difficult nor uncertain. For I may say that at Guy's Hospital for at least a quarter of a century there was no case of head injury examined —and such includes every case—in which there was not found some coarse brain lesion, readily visible to the naked eye; in which there was not some contusion of the brain surface, some laceration of the surface of the brain or of its substance; or more or less hæmorrhage upon or into the brain. In fact, concussion in a pathological sense, has been, in my experience, synonymous with contusion or laceration of the brain. Other surgeons have expressed the same opinion. Sir Prescott Hewett, thirty years ago, said: "In every case in which I have seen death occur shortly after, and in consequence of, an injury to the head, I have invariably found ample evidence of the damage done to the cranial contents," and Mr. Hilton wrote: "We ought to consider a brain which has been subjected to concussion as a bruised brain." Mr. Le Gros Clark also stated: "I have never made or witnessed a post-mortem after speedy death from a blow on the head where there was not palpable physical lesion of the brain." Neudorfer, of the Austrian army, has declared that he has never seen concussion, as so-called, since in all cases he has examined cerebral injury was found to exist. And Fano, a celebrated French surgeon, has also arrived at the conclusion "that the symptoms generally attributed to concussion are due, not to the concussion itself, but to contusion of the brain or to extravasation of blood." In fact, all authorities now agree that when death follows a severe shaking or concussion of the brain contusion, bruising, or laceration of the brain is invariably present, and that when this is not found the death is probably to be ascribed

to some other than a cerebral cause; and I shall be able, later on, to show that when death does not take place as an early result of the damage, and the patient either dies of some other affection or of some remote consequence of the injury, the same evidence of cerebral contusion is generally present. When, moreover, extravasation of blood upon or into the substance of the brain follows "concussion" or rather vibrating injury, it is to be explained in the same way—that is, by some injury done to the vessels of the brain itself, or to the venous sinuses within its membranes. When due to the bruising of the brain itself, the seat of injury is probably found on the side of the brain opposite to that of the cranium which received the blow; the bruising being brought about by what is rightly termed "contre-coup." Thus, a fall or blow upon the occiput is, as a rule, followed by some bruising of the anterior cerebral lobes; one upon the frontal region, by a bruising of the posterior parts of the brain; whilst lateral or vertical blows are felt more by the middle lobes. In severe vertical blows the base of the brain itself is bruised. The amount of extravasated blood depends upon the degree of force applied and the healthiness of the vessels in the injured part, diseased vessels easily giving way under a vibrating force to which the healthy would not yield. When the extravasation of blood is upon the surface of the brain, it is either within the cavity of the arachnoid or the meshes of the pia mater; and under each condition the blood gravitates to the base. When the extravasation of blood takes place into the structure of the brain, it may be found in any part of the cerebrum, cerebellum, pons varolii, or even in the ventricles, the extravasation rarely showing itself in the form of one large clot, but commonly in small and numerous spots of extravasation, which cannot be wiped away, as if from small vessels.

Thus I found in one case of so-called concussion, in which the fatal result took place sixty hours after the injury from changes brought about by the severe shaking of the brain, unassociated with fracture, that the brain was bruised all over, and that blood was effused at injured spots; the fluid in the ventricles was blood-stained, and the ventricles themselves ecchymosed.

In another case of death from "concussion," associated with fracture, the result of a fall, in a man aged 31, on the fifteenth day after the injury, in whom convulsions and coma supervened, a layer of blood was found universally diffused over both hemispheres, dipping between the convolutions, and passing downwards towards the base. The clot, which was shreddy and of a dull reddish-black colour, had evidently been effused for some days. The surface of the brain beneath the seat of injury was softened; and at its base, where it had been damaged by the contre-coup, similar changes had taken place. The vessels were healthy.

In a third case, where death followed from "concussion," and the vessels were diseased, multiple extravasations were detected after death throughout the substance of the brain.

A fourth case was that of a man (C. K.), aged 65, who came under my care with a scalp wound over the left half of the occipital bone and noisy delirium, having just previously fallen out of a truck on to his head. He had no paralysis or special head symptoms. The pupils were natural; the pulse was 70, and the temperature 99.2°. He became rational in twenty-four hours, but only remained so for a day, when noisy delirium returned, with refusal to take food. This condition lasted for fourteen days, when he sank, although food was regularly and carefully given by the oesophageal tube. After his death, on the sixteenth day from the fall, the anterior lobes of the brain, with the fore parts of the middle lobes, were found much bruised and covered with extravasated blood.

And in the fifth case—that of a man, aged 35, who came to Guy's on March 31st, and died on April 1st, 1882, about fifteen hours after the accident—blood was found extravasated over the whole surface of the brain beneath the membranes, and the brain itself was much ecchymosed in fine points. The third and fourth ventricles were full of blood-clot. There was laceration of the fornix and right optic thalamus, and no other lesion. The man had fallen on his head when jumping off a van, and given himself a scalp wound, exposing the bone, to the left of the occipital protuberance. He walked into Guy's, and complained only of headache and a feeling of sickness. He refused to stay in the hospital, and left. Having walked 200 yards, he vomited, and his friends gave him a "small soda." He then vomited again, and being unable to stand he was brought back to Guy's, where he was admitted insensible and comatose, and died, as already stated, fifteen hours after his fall.

When fissured fractures of the cranium complicate brain injuries, and these fractures are the

result of some diffused force, the cerebral mischief is not likely to differ from that which has been just described, although, as the force to produce a fracture may presumably be greater than that which fails to do so, the intracranial injuries may be greater from the cerebral vibration. In some cases the brain itself may, in addition, be bruised at the seat of impact. On the other hand, where the force which produced the fracture is concentrated, or the brain-case thin, there may be more of local brain injury at the seat of fracture, and less of distant mischief from brain vibration. And with the fracture there may be certain special complications, such as depression of bone with or without compression of the brain, injury to the dura mater, membranes, or brain from the fractured bone or external force, and extravasation of blood between the dura mater and the bone from rupture of the middle meningeal artery or some venous sinus. But all these are complications of, and additions to, the general injury. It must not be forgotten, however, that in the exceptional cases a fracture of the skull may take place from a concentrated local violence without producing any cerebral disturbance, particularly over the frontal region.

To prove still more conclusively that " concussion " of the brain means bruising or laceration of the brain, with more or less hæmorrhage, I propose now to bring before you the particulars of some cases which have been examined at Guy's Hospital after death, at more or less remote periods after the receipt of the head injury ; and which died from either an independent affection or some remote results of the injury. Such cases are not numerous, but they are valuable, at any rate, for my present purpose. I trust I shall not, therefore, prove wearisome in reading brief extracts of some of them.

Case 1.—Cerebral injury many weeks before death ; marked evidence of old bruising of the brain found. A powerful, middle-aged man was found by a policeman in the Borough Market sitting down in a fainting condition. The policeman gave the patient some brandy-and-water, and brought him to Guy's, where he soon became comatose and died. One of his friends said that he had received an injury to his head some weeks (?) previously. After death no external signs of injury could be made out, and there was no fracture of the skull. The anterior and middle lobes of the left hemisphere of the brain were

adherent to the base of the skull. The base of the anterior lobe showed brown discoloration from old bruising, and the middle lobe was so firmly adherent to the bone that it tore away. "These changes," wrote Moxon, who made the examination, " could not have been less than several weeks old, probably at least three months." There was a large effusion of recent blood-clot (two ounces) over the right side of the brain. There was no blood in the brain, and no lesion of its membranes. The viscera were healthy.

Case 2.—Cerebral injury complicated with fractured base of cranium thirty-eight days before death ; bruising of the brain. David E., aged 19, came under my care on June 24th, 1874, having fallen off an omnibus on to his head. He was admitted into the hospital, partly unconscious, with profuse bleeding from his *left* ear, which lasted for two days, and was followed for another eight days by the escape of a clear fluid (cerebro-spinal). On the second day after the accident the facial nerve became paralyzed. The man died on the thirty-eighth day from broncho-pneumonia, having lost all his brain symptoms, except the facial paralysis. On examination of the body a fracture was found in the skull, across the petrous portion of the left temporal bone, from the turn of the lateral sinus groove behind into the middle fossa, laying open the tympanic cavity. There was no trace of repair in the fracture. The olfactory bulb on the *left* side was gone, and the brain about it was bruised. The central parts of the brain were soft.

Case 3.—Cerebral injury complicated with fracture of the base of the skull twelve weeks before death ; evidence of bruised brain. (Preparation in Guy's Museum, 1084⁵⁵.) William B., aged 46, came under the care of Dr. Hilton Fagge in 1873, with vomiting after food and pain over the pylorus, which proved to be due to abdominal cancer. He had been an epileptic for four years, and during that time had been growing darker. Five weeks before admission, after a fall on the head from a ladder, he became insensible for a brief period, and blood oozed from his *right* ear, mixed with some watery fluid, which continued to flow for two days. All head symptoms disappeared, although he occasionally lost himself during his illness. He died from abdominal cancer. After death a fracture of the base of the skull was found, which crossed the petrous portion of the right temporal bone, and ran the whole length of the

meatus auditorius externus. The under surface of the two anterior lobes and of the points of the left middle lobe of the brain presented a tawny yellow discoloration, which was clearly the result of blood effusion at the time of the injury, and this, as usual, was more marked on the side opposite to that of the fracture. There was also a little superficial softening of the cineritious substance.

Case 4.—Cerebral injury six weeks before death, followed by abscess. William D., aged 30, was admitted into Guy's with pleuro-pneumonia, in a condition which soon ended in death, in March, 1873. Six weeks before his admission a beam had fallen upon his head, hurting him, but not producing any marked head symptoms or any bleeding from the ears or nose. Indeed, he had not given up work, but continued as an engineer's labourer for four weeks—that is, up to two weeks before admission, when he felt ill, shivered, and had severe headache; he also soon lost his taste for sweet things. When admitted he had no paralysis, only headache and drowsiness. His temperature was normal; he had no convulsions. After death the brain was found, on removal of the skull-cap, to be flattened, so that it appeared to have no convolutions. Its surface was discoloured in parts from what proved to be abscesses. A large and rather old abscess had burst into the hinder part of the right lateral ventricle, filling it with pus.

Case 5.—Cerebral injury complicated with fractured skull and spine, etc.; death ninety-one days later. (Preparation in Guy's Museum, 1084ᵐ.) George G., aged 27, came into Guy's Hospital, under the care of Mr. Forster, in July, 1882, having fallen or been thrown out of a third floor window. He was conscious, and complained of pain in his back. He had a scalp wound over the occiput, and evidently a fracture of the occipital bone. His lower limbs were paralyzed from a fractured spine. The patient died of pleurisy. At the necropsy a fissure was found in the right half of the occipital bone, which extended vertically across the root of the petrous portion of the right temporal bone towards the lesser wing of the sphenoid. In the posterior cerebellar fossa there were two offsets, one of which ran down to the foramen magnum. There were but feeble signs of repair in the fracture. The brain at the right anterior lobe was much bruised. There was blood between the bone and the dura mater, and on the inside of the dura mater there was much brown

pigment. The eleventh dorsal vertebra was fractured.

Case 6.—Cerebral injury six months before death, brought about by meningeal apoplexy; marked evidence of old cerebral injury. Patrick H., aged 41, was admitted into Guy's Hospital, under Dr. Pavy, in March, 1874, in a comatose condition, and soon died. He had been drinking for some days before, and had had a fit. Six months before, or thereabouts, after a fall down some stone steps, he was brought into the accident ward with bruise over the right mastoid process and discharge of blood from the right ear. He was partially insensible for ten days, and was stupid for some time after. His pulse ranged from 40 to 50. He left convalescent and returned to his work, at which he continued until he had the fit for which he was re-admitted just before his death. At the necropsy no injury of the cranial bones could be discovered. The dura mater over both sides of the brain presented on its inner surface a tawny red colour, apparently stained with old extravasated blood. That covering the left hemisphere was smooth, but that over the right was in part lined with adherent coagulum which in some parts was of a brownish colour, in others black. Some of it was evidently old. This was part of a large mass of coagulum which lay between the dura mater and the brain, on this side flattening the brain. The surface of the brain was so discoloured from staining with blood that the amount of clot was difficult of determination. The brain had evidently been deeply bruised at the time of injury at the inferior surface of both anterior lobes and the summit of the right middle lobe, particularly towards the back of the lateral surface of the right hemisphere, where there was an irregular fissure, with much tawny discoloration of the tissue. The lateral ventricles were healthy. The large effusion of blood clearly came from the rupture of the vessels of the softened part, and was probably in part due to alcoholic stimulants.

Case 7.—Cerebral injury thirteen months before death; marked evidence of brain injury. George L., aged 22, was admitted into Guy's Hospital under Dr. Habershon, in February, 1871, in a semi-torpid condition, having been found by a policeman in a fit after a day or more of drunkenness. He could be roused with difficulty, and when roused moved all his limbs. He had right facial paralysis. His pulse was 44; the pupils were contracted. He gradually sank. After death

no signs of injury of the cranial bones were discovered. The brain was flattened, and the anterior and middle lobes were adherent to the dura mater at the base, and the brain tore away when an attempt was made to remove it. The membranes of the brain at the base were very thick, and partly opaque white, partly of an ochre-yellow colour; this appearance was confined to the membranes. The aperture into the fourth from the third ventricle was closed by some recent lymph, and the brain bordering the channel was soft. The lateral ventricles were greatly dilated, the descending cornu on the left side projecting like a blister at the base of the brain. The left middle lobe was diffluent.

Case 8.—Cerebral injury two years before death from phthisis ; marked evidence of bruised brain. William C., aged 50, came into Guy's Hospital with phthisis, under the care of Dr. Moxon, in 1874, and died within a few days. He was a sailor, and had enjoyed good health up to two years previously, when he had a high fall, and was stunned, and bled from his left ear. The next day hot fluid came from the ear. He was laid up for three weeks, and when he left his bed he was giddy for nearly one year. He returned to his work, although deaf in the left ear. Five weeks before his admission he "caught a cold and cough," and died of acute phthisis. At the post-mortem examination no clear indication of fracture of the petrous portion of the left temporal bone could be made out, although there was a slight transverse line across the bone. There was an old bruise of the anterior lobe of the brain on the left side and slightly of the base of the middle lobe. That on the anterior formed an irregular hollow the size of a shilling. The grey substance was quite destroyed, and microscopically was found to present hæmatoidin crystals and compound granular masses.

Case 9.—Cerebral injury complicated with fracture of the base of the skull on the right side eight years before admission, followed by fits, chronic hydrocephalus, spinal curvature, emphysema, and hydrothorax ; marked evidence of old bruising of the brain. (Preparation in Guy's Hospital Museum, 1804⁵⁶.) David W., aged 30, was healthy until eight years before admission, when he fell off a ladder and hurt his head. Since then he had not been able to do hard work, and his memory had failed him. Five years ago he had a fit, and others had followed. He was admitted

under Dr. Wilks in 1876 in a fit, comatose and passing urine under him ; but he gradually recovered sensibility in a few days. In the hospital he had a fit, and both sides of his body were convulsed ; when consciousness returned it was found that the *left* side of his body had diminished sensation, but he said this had existed since the fall for eight years, and was gradually growing worse. He was fairly intelligent, and answered questions readily. His pulse was slow—38 to 40. He died of bronchitis. At the necropsy the brain was found to be flaccid. At the base of the right middle fossa, and over the roof of the orbit, there was a layer of brown pigment, also in the posterior fossa. The same pigment existed in patches over the base of the brain. There were fourteen ounces of fluid in the ventricles. The brain substance was healthy. The foramen magnum was altered in shape, and narrowed antero-posteriorly, the transverse diameter being much larger than the other. There was fracture of the right side of the base of the skull. The spine was curved at the sixth dorsal vertebræ towards the right. The cord was healthy. The lungs were emphysematous ; fluid existed in the left chest.

Case 10.—Cerebral injury complicated with fracture of the skull nine years before death ; evidence of old as well as of recent brain injury. Michael L., aged 50, came under Mr. Cock's care on December 29th, 1867, and died on January 7th, 1868. He had fallen twenty feet on to the side of his head, and was admitted with a scalp wound and depressed fracture of the left of the vertex. He was unconscious on admission, but not paralyzed, and was never clear enough to give a history of his accident. One of his friends stated that he had had an injury to his head nine years previously. After death the cranial bones were found to be thin. On the occipital base, about the left part of the groove for the torcular Herophili, were *fine* rough elevations, and here the dura mater was very adherent. About this part there was an old depressed irregular fracture of the bone, the bone being now united to the rest. Within the skull there was an elevation corresponding to the external depression, and its edges were bevelled off. There was no sign of any old external wound over the fracture, or of blood between the bone and dura mater ; but on opening the latter about an ounce of liquid clotted blood was found effused over the left vertex, corresponding to the recent scalp wound. The right anterior lobe of the

brain was adherent over the orbit, and the brain here was discoloured brownish yellow, as from old hæmatoma, this part corresponding to the spot of contre-coup from a blow on the left back of the head.

Case 11.—*Cerebral injury complicated with fracture; evidence of brain bruising, recent and old.* Michael W., aged 53, came into Guy's Hospital, under Mr. Cock, in February, 1867, and died in two days. He was found in the street insensible, with a wound on the back of his head, and bleeding from both ears, but chiefly the left. He died comatose. At the necropsy there was clear evidence of a heavy blow having been given over the right mastoid process, the bone at this part being fractured. There was no blood between the bone and dura mater at this part, but there were about two ounces between the dura mater and the brain. There were *recent* contusions on the anterior and middle cerebral lobes, and *old* yellow discolorations of both middle lobes. The right one was firmly adherent to the middle fossa outside the foramen ovale. Lining the dura mater of the right middle fossa of the base of the skull there was a thin membrane, easily separable; and this was thickest where the brain was adherent. The right middle lobe was more deeply bruised than other parts, where it was fixed to the fossa.

Case 12.—*Compound fracture of the cranium, with marked brain symptoms, occurring three years and a quarter previously; necrosis of the inner table of the skull; removal of the bone by trephining; death from phthisis; evidence of bruised brain and extravasation of blood.* Conrad H., aged 45, came under my care in January, 1879, with a discharging sinus on the right side of the occipital protuberance, communicating with the interior of the cranium, which had been the result of a compound fracture of the skull he had sustained three years and a quarter previously. The injury was brought about by a blow from a large stone, which stunned him, and he remained unconscious for seventeen days. He was abroad at the time and had no treatment. About one year afterwards some pieces of necrosed bone were taken from the vertex of the skull, the result of a second fracture he had received at the same time. He had then some slight weakness of his left side. For the last year the sinus had discharged freely, and he had suffered from much headache. Finding on examination that some necrosed bone could be felt within the cranium, in the

direction of the internal occipital protuberance, I trephined the bone with an inch trephine, and enlarged the opening with Hoffmann's forceps. Having done this, I removed numerous elongated fragments from the inner table of the skull, corresponding with the lateral and longitudinal sinuses. The dura mater within was thickened, and covered with granulations. The operation gave much relief to his head symptoms; but his lung trouble slowly extended, and destroyed life five months subsequently. After death the repair of the base of the skull was found to be complete, but the bone was thick and irregular. The opening in the cerebellar fossa was only partially closed. The dura mater corresponding thereto was thick. The torcular Herophili and sinuses were healthy. The dura mater covering both cerebral hemispheres was tawny with old extravasated blood; indeed, a thin membranous film could be stripped off its inner surface. The summits of the middle lobes of the brain at its base showed some tawny erosion, evidently the result of former bruising. The lungs were extensively diseased.

The evidence I have thus laid before you of the pathological conditions of the brains of those who have suffered from what has been so long known as "concussion of the brain," will, I trust, be deemed sufficient to convince such as may be in doubt that they are really examples of cerebral injury; that term meaning cerebral bruising or laceration, with more or less hæmorrhage upon and into the substance of the brain or its ventricles; the amount of injury of every kind varying in degree in each case, in some it being very slight, and in others severe.

You will likewise have probably observed—what the details of the cases I have brought before you so forcibly demonstrate—the lasting character of the changes which the brain may have undergone as a result of injury, and the evident slowness with which nature performs in this organ her reparative work; for, in some of the cases quoted, years had passed after the injury had been received, and yet marked evidence of its former existence was still present. Indeed, such evidence as I have laid before you, of necessity, suggests the questions : Is a bruised brain ever thoroughly repaired? and, are not the changes which the injury may have brought about fixed and permanent? it is to be regretted that an answer to these questions can only be given in an unfavourable form; and that, whilst we may be hopeful as to

this complete repair and recovery from a slight injury or bruise ; we are bound to regard graver cases in a more serious light, and to deal with them accordingly, since what evidence we possess seems to show that, when any portion of the brain has been severely or moderately bruised, it has been permanently injured. This evidence also dovetails in with the general experience, which tells us not only of the presence of physical head symptoms, but that of the mental and moral characters of men are often permanently altered by a head injury.

With these facts and conclusions before us, am I, therefore, wrong in assuming with some confidence, that you will see with me the expediency of combining with the term "concussion" that of "injury," and of describing such cases in the future as those of bruising or injury of the brain from concussion ? The term "concussion" by itself is vague and delusive, whilst that of "bruising" and "injury" is clear and true, and conveys at once a meaning, the force of which cannot be misunderstood. The word "concussion" later on may be dropped, and the simple term "bruising" retained. With this starting-point, it would naturally follow that fractures of the skull in all their varieties; hæmorrhage into the cranium in all its forms; and compression of the brain, however brought about, will be regarded as complications of the one common and essential factor, cerebral injury, and not, as now, be regarded as separate and individual troubles to be dealt with independently. And even scalp wounds, the result of external violence, would assume a position in the surgeon's mind they ought to possess, but have not yet attained; and consequently receive the attention to which they are entitled, not so much perhaps on their own individual account as simple wounds, but as wounds mostly brought about by direct violence applied to the cranium, and consequently liable to be complicated with some contusion of the cranial bone or intracranial injury.

Up to this time my observations have been confined to the elucidation of the first of the two main clinical points which have had a common bearing upon all cranial injuries, and to which I drew your attention at an early period of this lecture, namely, "that all injuries of the head should be estimated *primarily* with reference to the amount of damage the cranial contents have sustained;" and I trust I have demonstrated with sufficient clearness that a cerebral injury of some

kind is the one common factor. I propose now, therefore, to pass on and consider the treatment of cranial and intracranial injuries, and, with the light which the above conclusion throws upon the whole subject, see what bearing it ought to have upon the second clinical point to which attention has been drawn, viz., "that injuries of the head should be estimated *secondarily* with reference to the risk of the cranial contents becoming involved"; and it should be remembered that this risk is one to which every degree and variety of cranial injury is liable. In even such an apparently simple accident as a contusion of the head, whether with or without a scalp wound, the fear of this secondary danger ought not to be overlooked ; indeed, it ought always to be held in view, and considered possible, for I imagine there are but few surgeons who have not been called upon to treat examples of scalp wounds, or patients who, having had cranial blows and being supposed to have been cured, have, on going to work or moving about, or after some indiscretion of diet, complained of headache, restlessness, giddiness, nausea, or even vomiting, with more or less febrile disturbance—all these symptoms being those of cerebral irritation, or the first step of inflammation. Or possibly the patient has complained only of local pain at the seat of injury, and the surgeon on examination finds some swelling of the soft parts over the bone or some change in the appearance of the wound, its healthy granulating surface having assumed a pale, flabby condition, always suggestive of an early ostitis or periostititis. "Acceleration or hardness of pulse," wrote Percival Pott, "restlessness, anxiety, and any degree of fever, after a smart blow on the head, are always to be suspected and attended to. . . . . When there is a wound, it will for a time have the same appearance as a simple wound. But after a few days all these favourable appearances will vanish ; the sore will lose its florid complexion and granulated surface, and become pale, glassy, and flabby ; instead of good matter, it will discharge only a thin discoloured sanies, and the pericranium will separate from the bone. The first appearance of alteration in the wound immediately succeeds, the febrile symptoms increase, the sore becomes worse and worse. . . . Through the whole time, from the first attack of fever to the last and fatal period, an attentive observer will remark the gradual alteration of the colour of the bone, if it be bare. At first it will be found to be whiter and more dry

than the natural one, and as the symptoms increase the bone inclines more and more to a kind of purulent hue of whitish colour."

These extracts I have taken from the works of Percival Pott, who first drew attention to this danger of bone inflammation as a result of cranial injury a century ago, but possibly made too much of it. He regarded it as the chief element of danger in all cases of contusion, scalp wound, or fracture, and not only trephined the skull when ostitis existed, but laid it down as a rule that "perforation of the skull is absolutely necessary in seven cases out of ten of simple undepressed fracture." The operation for trepanning was called for in these simple cases "to *prevent* the effects of inflammation, detachment, and suppuration of the dura mater, and consequently the collection of matter between it and the skull." From this over-estimate of the value and necessity of the operation of trephining in cases of fracture, and the comparative rarity of cases of abscess between the bone and dura mater as a direct result of contusion, surgeons have been prone to treat too lightly the risks of a secondary ostitis following bone injury, whether complicated or not with scalp wound or even with fracture, and consequently to neglect what was good in Pott's teaching. As a result, I feel sure that many lives are lost yearly, and that many narrow escapes from death occur. I have the notes of some fatal cases of the kind before me, which I have taken from the post-mortem records of Guy's Hospital. In four of these the immediate cause of death was pyæmia, and such a result was probably brought about by the inflammation of the venous channels of the diploë of the injured bone. I will not weary you by reading all the details of the cases, but will lay before you their chief points.

Case 13.—Scalp wound; ostitis; necrosis; pyæmia. T. G., a man aged 22, having fallen from a height, came into Guy's Hospital with a scalp wound and a compound fracture of his leg. He died from pyæmia. The scalp wound had apparently healed, and the man with his compound fracture seemed to be doing well in all ways, when on the tenth day headache and febrile disturbance appeared, followed by swelling in the seat of cranial injury, reopening of the wound, and suppuration of the parts covering the bone, from which the pericranium had loosened. Indications of pyæmia soon appeared, but no general head symptoms, and he died in two weeks. At the

necropsy, the anterior half of the parietal bone beneath the scalp wound was of a whitish colour, bare for a space of three by two inches, and apparently dead. The necrosis extended through the bone, and on the surface of the dura mater beneath the bone there was lymph with pus, as also on the arachnoid surface. The brain was healthy. There were pyæmic abscesses about the body.

Case 14.—Scalp wound; ostitis and necrosis of the injured bone; meningitis; pyæmia. Eliza S., aged 40, came into Guy's Hospital with a large scalp wound over the right parietal region, and exposed bone. The injury was the result of a fall off an omnibus, which for a few minutes stunned her. The next day she complained of headache, and on the fourth day it was incessant. On the ninth day sleeplessness and indications of fever appeared; the wound also became unhealthy. On the twenty-first day there were rigors, and on the twenty-fifth some hemiplegia on the left side, for which the operation of trephining of the right parietal bone was performed, and some fœtid pus evacuated from beneath the bone. The dura mater beneath the bone was velvety. Convulsions soon came on, and death in a week. At the necropsy, the whole thickness of the right or injured parietal bone was found to be dead, and the dura mater beneath discoloured. The surface of the brain corresponding to the dead bone was covered with pus. Pyæmic abscesses were present in the lungs and liver.

Case 15.—Scalp wound; ostitis of the external table, fracture of the inner table; pyæmia. E. N., aged 18, having received a scalp wound to the right of the vertex of his skull from a falling bucket, came to Guy's Hospital without any head symptoms, and had his head dressed. The wound healed rapidly without pain or trouble in about ten days, when some local swelling appeared, followed in two days by fever and rigors. In this condition he was admitted into the hospital, where pain and swelling of the right elbow-joint appeared, and later on chest symptoms, which proved fatal three months after the primary accident. After death the outer shell of the parietal bone beneath the scalp wound, to the extent of an inch and a half by one inch, was necrosed, but it had not been fractured. The inner surface of the corresponding area of bone was fractured, the fracture lying in the long axis of the oval necrosed piece. The fracture was a mere fissure, slightly

starred, and its edges were perceptibly raised. A little stringy lymph hung about the bone. Over the dura mater corresponding to the fractured bone there was a yellowish patch of lymph. The brain was healthy. There were pyæmic abscesses in the lungs, liver, and elbow.

Case 16.—Scalp wound; exposed and inflamed bone; death from bronchitis. A man, aged 23, having been jerked from the shaft of a cart, received a blow and a wound over his left temple which exposed the bone. There were no head symptoms. The wound was carefully dressed antiseptically, and for a week everything went on well. At the end of that time his temperature went up to 104.6°, and the glands of his neck commenced to enlarge. On the tenth day he had paralysis of the left facial nerve, and bronchitis set in, which quickly destroyed the patient, three weeks after the injury. At the post-mortem examination, two square inches of the left or injured temporal bone were exposed. The bone was dry and discoloured, but there was no obvious necrosis. The diploë of the bone in the line of section of the calvaria on the left side was more vascular than that on the right side. The brain and dura mater were healthy. The bronchial tubes were filled with viscid mucus. The other organs were healthy.

Case 17.—Scalp wound; necrosis of bone; abscess in the brain. A male child, aged 3, six weeks before death fell on his forehead, causing a wound one inch above the left orbit. No head symptoms were induced by the fall, and for three days all seemed to be well, when he fainted in his mother's arms. He was then brought to Guy's Hospital, and requested to be left; but to this the mother objected. A few days later the child again fainted and vomited, and as the vomiting persisted the mother brought her child into the hospital. At that time, sixteen days after the accident, the wound had healed, and the scar was adherent to the bone. The child vomited daily, and his temperature was just above normal. A week later, without any important change in the symptoms, the child had a fit which lasted an hour. During the fit the right leg was rigid, and the arm with the fingers and thumb became spasmodically flexed. The left limbs also moved spasmodically. Temperature, 106°. In two days the child died. After death the frontal bone at the seat of injury was covered with purulent lymph beneath the pericranium. Beneath this the bone was rough

and ulcerated. The inner table over a space the size of a sixpence was opaque yellow. When sawn through a piece of the outer table was loose and necrosed. The dura mater at this spot was adherent to the brain. There was a little pus on its outer surface. The left frontal lobe of the brain beneath this spot was soft, swollen, and fluctuating, and when cut into was found to contain a large abscess of the size of an apricot, which had a distinct lining membrane. There was no red softening.

Case 18.—Scalp wound; necrosis of bone; abscess of the brain. A middle-aged woman, three weeks before her admission into Guy's Hospital, received a scalp wound from a blow upon the forehead. She continued at her work for two weeks, and it is said without any head symptoms, when she became comatose, and died in a week. She was brought to the hospital in the dying comatose stage. At the post-mortem examination a sloughing wound was found to occupy the centre of her forehead, and beneath it bone was exposed. The surface of the bone was yellow and dead, but not depressed. The area of dead bone was separated from the living by a shallow groove. The inner surface of the bone was blackish; the diploë was full of pus. There was chocolate-coloured pus beneath the dura mater of the injured part, and the brain beneath was suppurating, the abscess burrowing backwards.

Case 19.—Contusion of cranium; Pott's "puffy" tumour; trephining; meningitis; death; bruised brain. W. L., aged 46, was admitted into Guy's Hospital, under the care of Mr. Howse, on December 13th, 1882, fifteen days after having been knocked down by a cab and stunned. He was taken home after the accident, unconscious, and remained in that state for three hours. He then vomited and brought up blood. The next day he became drowsy; and four days later he had a convulsion, which was followed by others for three days. He then became delirious and unconscious. When admitted he was in a low typhoid state, constantly muttering and picking at the bed-clothes. Temperature 100.4°. There was no paralysis. A swelling was found over the vertex of the skull. This was cut upon, and blood was soon effused beneath the pericranium, which readily peeled off the bone. The bone was bruised and yellowish. The inch-trephine was used. The dura mater, which bulged into the opening and was covered with lymph, pulsated.

Nothing more was done, except that the wound was dressed, and cold applied to the head. The delirium, however, continued, and gangrenous pneumonia set in, which proved fatal on January 13th, thirty days after the accident. After death the base of the brain was found to be bruised by contre-coup, and there was diffused meningitis.

The cases I have quoted were all examples of ostitis and necrosis, the result of a contusion of the bone associated with scalp wound. I could give as many more associated with fracture if they were needed, and they would all tell the same tale. In none of the cases were there any symptoms of brain injury after the accident, and in most the symptoms did not appear for a week or ten days or a fortnight afterwards. In all, the mischief which produced death had clearly originated in the bone. In none of the cases had much care been employed to guard against the secondary mischief which took place, and which led on to a fatal issue; and it may reasonably be thought that, if judicious treatment had been applied from the receipt of the accident, no such result would have been recorded. The conclusion is, therefore, clear, that all scalp wounds which lead down to bone should be dealt with, for at least a fortnight or three weeks, with much care; and that such cases should, if possible, be treated as in- and not out-patients of hospitals for that time. For it is not the wound treatment only which calls for care, but the patients should be kept quiet, and given a simple unstimulating diet. Stimulants of all kinds should be forbidden, and meat allowed in very limited quantities. If, at the end of two or three weeks, or thereabouts, no local or general symptoms appear to suggest mischief, the duties of life may be gradually recommenced. But even then a caution should always be given to observe care.

When local symptoms appear, such as have been described, a free incision down to the bone where no wound exists, or a free separation of the pericranium where there is a scalp wound, always does good. And should any symptom appear or persist which even suggests any intracranial complication, the operation of trephining should at once be resorted to. "The spontaneous separation of the pericranium, if attended with general disorder of the patient, with chilliness, horripilation, languor, and some degree of fever, appears to me," says Pott, "from all the observations I have been capable of making, to be so sure and certain an indication of mischief underneath, either present

or impending, that I shall never hesitate about perforating the bone in such circumstances. . . . When there is just reason for supposing matter to be found under the skull the operation of perforation cannot be performed too soon; it seldom happens that it is done soon enough. The perforation sets the dura mater free from pressure, and gives vent to collected matter, but nothing more. The inflamed state of the parts under the skull and all the necessary consequences of such inflammation calls for all our attention fully, as much afterwards as before; and although the patient must have perished without the use of the trephine, yet the merely having used it will not preserve him without every other care." The prevention of this fatal trouble is, however, the more important point to emphasise; and for that purpose I bring the subject before you. Its early treatment may be beneficial and successful; its later treatment cannot be said to be so. Let us, therefore, teach the necessity of keeping patients with all but minor scalp wounds, and with those even where the bone is exposed, quiet and un-stimulated for some weeks after the receipt of the injury, where it can be done, and by so doing give nature an opportunity of repairing the mischief in the bone, which, though unseen, may reasonably be expected to be present after the application of a force sufficient to produce a scalp wound or a more severe injury. When a blow upon the head is known to have produced a fracture the case is likely to be treated carefully; whereas, when no such fracture can be made out, and there is little or no external evidence of injury, the same care is not likely to be observed, although in both cases the violence which had been employed may have been equal. Yet in both cases the dangers of cranial ostitis from contusion are about the same. The fact, I am sure, requires to be emphasised, that cranial contusions, whether associated or not with fractures or with wounds, are always matters of serious importance, and as such should be treated from the first.

I will now pass on to consider the treatment of head injuries in the light of the view I am now advocating, for I am under a strong impression that such a view cannot do otherwise than have an important influence in rendering treatment more simple and intelligible; since if in every grave, or, indeed, apparently complicated example, associated with more or less complete paralysis of at least one of the brain functions, such as is indicated by

unconsciousness, the surgeon recognises to the full the force of the fact that the brain as a material organ is bruised or otherwise injured, a line of treatment is likely to be at once suggested which can best favour the restoration of the injured part towards health.   Amongst the means which would probably find favour, physiological and mechanical rest would stand foremost, with the administration of nourishment simple enough to maintain the normal powers and help repair, and not stimulating enough to excite action.   Everything in the form of alcoholic stimulants or solid meats would be forbidden; and this line of treatment would, moreover, be maintained for weeks, and possibly for months, the severity of the injury and the primary symptoms forming the surgeon's best guide to a decision.   This careful line of treatment would also be adopted under the wholesome dread of exciting, by sins either of omission or commission, the one common complication which the surgeon should ever have before him—namely, an inflammatory action in the injured organ.   For experience speaks in no feeble terms that this action is readily started and with difficulty quelled, and that it is by such inflammatory changes in the injured brain that most head cases, simple or severe, are brought to a fatal termination.   Mr. Hilton recognised this necessity nearly thirty years ago, for he taught that "recognised lesions of the brain and its membranes, associated with blows upon the head (whether the cranium be fractured or not), do not generally, or as a principle of treatment, obtain that extent of mechanical rest which is consistent with the expectation of perfect and complete structural repair.   This error in the treatment of such cases is one of the chief sources of the diseases of the brain and its membranes which are met with in practice. . . . In cases of injury of what may be called the coarser structures, with more simple functions attached to them, we see that without perfect restoration of the structures, their functions are not efficiently performed, and if used too early and too much they become painful, and assume a chronic inflammatory condition. Such soft parts require weeks or months for their repair.   Surely, then, we ought not to deny the necessary and proportionately much longer time for the repair of the more delicate brain tissues; a repair, be it remembered, which cannot be accomplished by any direct aid from the surgeon, but only by nature herself employing her chief agent—"rest." It is a pleasure to me to be able to quote these apt sentences, framed by a former teacher and colleague upon this important subject —although many years have passed since they were uttered,—to support the views it has been my privilege to bring before you—views, I may say, that I have for long taught at Guy's, and, have reason to believe, with some advantage.

Should symptoms of intracranial irritation or inflammation show themselves, they should be dealt with actively, as, from the nature of the brain and its membranous coverings, the process once started soon spreads.   In the early stage the application of cold to the head by means of a Leiter's metallic tube is the most efficient local, and free purgation the most effective general, means, with a very low diet.   If the inflammatory action is great, a free bleeding from the jugular vein, or from the arm, is strongly to be advocated, and this operation may, in many cases, be repeated with much advantage.   I am convinced I have saved some lives by this treatment.   In chronic cases the value of mercury, taken internally, cannot be doubted.

These, then, are the common lines upon which the treatment of the common factor of all cranial injuries, simple or severe cerebral injury, should always be based; and they should likewise form the lines of treatment of all its complications. Thus, if a simple fracture of the vertex, base, or of both, complicates the case, the treatment is the same.   The cerebral injury needs the surgeon's care, and not the fracture, which will take care of itself.   A cranial fracture will heal in the same way as other fractures, but it will take a much longer period; and fractures of the skull are apparently amongst the slowest.   In specimens 1084[82] (eighty-four days), 1084[55] (ninety-one days), and 1084[55] (eight years), in Guy's Museum, this point is indicated. The fractures of the skull will, at any rate, probably heal sooner than the cerebral injury will be repaired.   The treatment for the latter will consequently have to be continued after the fracture has healed.   If the fracture be but *slightly depressed*, whether simple or compound, and it appears only as a *fissure*, the case had probably better primarily be left alone, and dealt with secondarily on the smallest indication of cerebral trouble; for in these cases there is rarely comminution of the inner table, and consequently nothing in the form of bony spicules to fret and irritate the dura mater, and thus help forward a meningitis.   To trephine in order to elevate this form of fractured fissure would therefore be to add

another danger to a case in which the form of cerebral injury to all already exists. If the fracture be *depressed, starred, or comminuted,* whether simple or compound, the elevation of the depressed bone should be the rule of practice, and the removal of all the splintered fragments of the inner table carefully carried out; the object of the operation being more to take away what, if left, must irritate the dura mater, and so add to the existing harm, than to relieve the depression. This operation should be performed as much in simple as in compound fractures, for the condition of the bones is the same in both; and with our modern treatment of wounds the danger of the operation in simple fracture is not materially increased. In *punctured fractures* the operation of trephining, undertaken with the object of removing the broken and displaced fragments of bone, should be a rule of practice never to be deviated from. The depressed and comminuted inner plates of bone to a certainty, if left, at a late if not early period of the case, irritate the brain and its coverings, and so set up an encephalitis.

How far the presence or absence of what are called brain symptoms should influence a surgeon in his decision as to surgical interference in the different forms of depressed fractures of the skull we have been considering has been much argued. For the surgeon is quite unable in bad cases of cranial injury to differentiate symptoms, and to say how far those that are present in any individual case are due to the common factor—cerebral injury—which resulted from the force that produced the fracture, or how far they are caused by the depressed and fractured bone. But I am not sure that this is a point of much practical importance, for in a bad case of fracture of the skull cerebral injury is probably already severe, and the operation of elevating depressed bone and of removing comminuted fragments is not likely to aggravate the trouble; whereas, in a less severe example, in which the cerebral injury is likely to be less serious, the existence of depressed bone and of comminuted fragments must act injuriously, and should consequently be removed. Under all circumstances, it is consequently the surgeon's duty to remove whatever sources of trouble the presence of a depressed fracture may bring to an already serious case of cerebral injury.

The operation of trephining, or of elevation of bone in depressed fracture, is called for more with the object of removing from the brain what

may or will be sources of local irritation rather than with any view of removing the effects of the depressed bone; for it is well recognised that, *per se,* a large area, and a considerable amount, of depressed bone is required to bring about symptoms of compression in an otherwise uninjured brain; and, it is also well known that a considerable extravasation of blood upon the surface of the brain, probably five or six ounces, whether between the bone and dura mater, or in the cavity of the arachnoid, is required to bring about marked evidence of its presence, in the form of paralysis from compression.

With these remarks on head injuries as a class, I now close my lecture. The opinions I have expressed are not new, for they are what I have taught for years, and the practice based upon them I am convinced is the only sound one. The material I have laid before you I originally laid before the members of my college, in 1888; but it is so suggestive, if not demonstrative, of the action I want you to adopt that I have taken the present opportunity of again bringing it into public notice.

[At the conclusion of the lecture, Mr. Bryant expressed his regret that three cases illustrative of the contentions above reported were not in attendance, but owing to circumstances entirely beyond his control it was impossible to have the three subjects present at the same time, and as one subject would have been of no utility, he had thought it better to give the lecture without any practical demonstrations.]

Cold Powder.—

R   Camphor   ...   . $\bar{3}2\frac{1}{8}$
    Ammon. carb.   ...   ... $\bar{3}$iss
    Pulv. opii   ...   . $\bar{3}3\frac{1}{4}$
    Potass. sulph. ...   . $\bar{3}$iv
    Powd. ipecac. ...   .   gr.ss

M. Ft. pulv. Sig.: Used whenever Dover's powder is indicated, and to alleviate cough of phthisis and pneumonia.

(*St. Louis Courier of Medicine.*)

Sphacelotoxin.—Obtained from ergot. Yellow powder, insoluble in water; soluble in ether, chloroform, alcohol, and alkaline fluids. Effects similar to ergot, manifested in several minutes, attaining maximum in half an hour. Dose, gr.$\frac{4}{5}$ to $1\frac{3}{4}$.—(*Nouveaux Remèdes*).

A CLINICAL LECTURE

ON CASES OF

SAVILL'S DISEASE, PARAPLEGIA, PLEURITIC EFFUSION, AND HEART DISEASE.

Delivered at St. Mary's Hospital, Oct. 17, 1894, by

D. B. LEES, M.D., F.R.C.P.,

Physician to the Hospital and to the Hospital for Sick
Children.

A CASE OF SAVILL'S DISEASE.

GENTLEMEN,—Some of you will probably remember that in July last I showed you a man, aged 53, suffering from an advanced stage of chronic Bright's disease, with extremely high arterial tension, on whose skin a peculiar eruption had appeared. It consisted of groups of papules tending to coalesce into erythematous areas, and was found on his lower limbs, on his upper limbs, and on his back.

It reminded me very strongly of four cases which we had in this ward in the year 1891, at the time when an epidemic, numbering 163 cases, of a similar eruption occurred in the Paddington Infirmary. That epidemic was carefully investigated by Dr. Savill, and he published his results in this excellent monograph which I showed you. He studied the disease not only from its clinical but also from its pathological and bacteriological aspects, and detected a diplococcus which appeared to him to be characteristic of the disease and to have certain properties which differentiated it from all other similar cocci. I showed you this pamphlet of Dr. Savill's at the time, with the excellent photographic illustrations which it contains of patients suffering from various stages of this curious skin disease.

I read to you also the description of my own four cases, in a letter to the *Lancet* of August, 1891, and I ventured to prophesy that if we were right in our diagnosis of this particular case we should before long have other cases of the same kind in the hospital. That prophecy was verified, for within a few days after the death of the patient to whom I referred another patient was admitted with renal disease, who developed the same sort of rash within three or four days of his admission into the adjoining ward, so that whether he caught it in the hospital, or whether he brought it in with

him, we cannot determine, but perhaps the latter is more likely. Since then I believe there has been a third case in the isolation ward, under the care of one of my colleagues, and probably a fourth, so that we have again had a small epidemic. Now, as we had been free from this peculiar affection during the years 1892 and 1893, I felt a little hesitation in arriving at the diagnosis, and I therefore asked Dr. Savill, who had studied the matter so carefully, to be good enough to come down and see this particular case. He did so, and at once confirmed the diagnosis; and when the patient died, not very long afterwards, he was good enough to attend at the post-mortem, and, having provided himself with means for bacteriological research, he succeeded in obtaining pure cultures of the micrococcus from the blood of the patient's heart. I have here a culture tube which Dr. Savill has been good enough to send, and which, if you hold it up to the light, will show the growth of the micrococcus. You see it is growing entirely on the surface of the agar. It is an aërobic parasite. I will read you a short extract from the letter which Dr. Savill has kindly sent me: "The post-mortem was made about thirty hours after death. The great vessels at the root of the heart were tied and removed with the heart, and the whole quickly plunged into a solution of perchloride of mercury (1 in 1000). A sterilized knife made an incision into the left ventricle, a platinum point was plunged in, and four culture tubes (two of gelatine and two of agar) were inoculated from the blood. Three of these tubes gave a pure culture, having these characteristics:

1. A *thin* white growth, with slightly raised crenate margins.

2. Aërobic; it will not grow in absence of air.

3. It does not deliquesce gelatine, and is thus differentiated from staphylococcus albus, and nearly all saprophytic and putrefactive organisms.

4. Microscopically, oval cocci measuring about one micro-millimetre, generally arranged in pairs.

Turning again to Dr. Savill's monograph, we find in it a micro-photograph which shows very well these cocci, and with a lens you can easily see that they are arranged in pairs; it is therefore a diplococcus. Thus it seems that Dr. Savill has traced the disease to its origin, and his results have been confirmed by Dr. Russell. The nature of the changes in the skin in this peculiar disease has been studied by Dr. Echeverria at Hamburg, and he is on the point of issuing a paper on the

subject, of which I have seen an advance proof, in which he maintains that there are certain peculiar changes, especially in the nuclei of the prickle-cells of the epidermis, which differentiate this disease from eczema, and from other diseases which it more or less resembles, so that if these observations be confirmed there can be no doubt that we are dealing here with a peculiar and special disease, to which attention has not previously been drawn, and it is clear that we ought for the future to call it "Savill's Disease."

With regard to its features during life, they are very fairly shown by this coloured drawing, which Dr. Savill has had made from this very case. You will see in this drawing that the back of the patien' is covered to a very large extent with an eruption, and that the most recent parts of this eruption ar·· over the higher part of the back; it began on th·· legs, and gradually travelled upwards, so that on the upper part of the back it is most recent, and there we find that it appears in small papule· which have a great tendency to become clustered into groups. Such groups are occasionally ver) nearly rings, but more generally definite groups, and after a time these groups tend to coalesce. If, next, you look at the lower part of the back, you will see there a large erythematous area on each side of the spine, symmetrically, and over these areas the disease is beginning faintly to desquamate. Thus we have indicated all the characteristic features of this eruption. It commences in these grouped papules, then forms erythematous areas, and finally tends to desquamate. Sometimes this is very marked, the epidermis coming off in large flakes as in the desquamation of scarlet fever, and occasionally in much larger scales, as is well shown in the photographs in Dr. Savill's pamphlet.

I do not know that we can say that in this case of ours the fatal result was due to Dr. Savill's diplococcus, for the condition of the patient was already desperate, and I was surprised that we were able to keep him alive so long. But there is no doubt that this microbe is specially prone to attack patients suffering from renal disease, and that such cases frequently die. Cases of dilated heart, in many of which there is no doubt some morbid condition of the kidneys, also suffer severely.

The mortality from this affection among patients of these two classes is very high, and the disease is decidedly dangerous to patients in the later half of life. It may attack also the young and strong, but to them it usually causes little inconvenience.

One peculiarity about it is worth remarking, that whether the eruption be slight or severe, there is usually an absence of pyrexia.

A CASE OF PARAPLEGIA.

This patient, Gentlemen, I show you this afternoon, because his case illustrates some points which we have studied in my systematic lectures on "Diseases of the Spinal Cord," in which we have been discussing the various methods of conduction in the cord, the reflexes, and the circumstances which increase or diminish such reflexes.

Now this patient has here in his lower dorsal region an undue prominence of two or three vertebræ; he is suffering from spinal caries. Without going into the case at all exhaustively I want to draw your attention to one or two points about the condition of the legs. In the first place, with regard to the condition of the reflexes, you will remember that I said that any compression or disease affecting a transverse section of the spinal cord above the lumbar enlargement has a tendency to increase the reflexes in the area below, and I will just show you how true that is in this particular case in the muscular reflex, in the extensors of the knee. If I gently raise his thigh, and give a slight tap on his patellar tendon, instantly there is an extraordinary jerk, and the same thing takes place on the other side. The knee-jerks are very greatly increased. If I place my finger on the patella and gently depress it downwards and then quietly tap my finger, I get again a sharp contraction, or what is known as a supra-patellar jerk; the same thing occurs in the other leg. If I depress the patella more forcibly I get a clonus, not only one but several muscular contractions, following one another in rapid succession, and it is a point of interest in regard to this patient that even the very act of forcibly depressing the patella sets up a clonus in the extensors without any blow, the muscular "tone" being so much increased that even so slight an increase in tension sets up these rhythmic contractions. I have in my lectures described "ankle clonus," and you can see it brought out here. You notice I have only to press up the sole of the foot, and this repeated motion is set up and maintained with very considerable pertinacity and perfect regularity while it lasts, and the same symptom occurs in the other foot.

The next point which I want to mention is with regard to the sensory paths, and you will remember that in studying the different kinds of sensation we found that these are not always affected alike in diseases of the spinal cord. They travel by different paths in the cord, and probably they are subserved by different nerves; thus, one may be affected when another escapes.

And first we will deal with the sensations of touch and pain. You notice that on applying the usual tests, there are some points where the patient cannot feel the touch of my hand, but where he feels at once the prick of a pin, thus showing that over that spot there is loss of tactile sensation, but no loss of painful sensation, and indicating that the two different sensations are either conveyed by different nerves, or that they have different paths in the cord, one of which is destroyed and the other is not.

And next, we will try the temperature test, and by means of a hot spoon and of ice see whether he is able to distinguish heat and cold, and to tell the one from the other. You see his conclusions are often erroneous, and that in many places he cannot detect the difference between heat and cold, and in some mistakes the one for the other.

There is one sense still remaining, and that is derived from the muscles; this we test by the patient's knowledge of the position of the limbs. But while we apply this test, we must remember that there is always this fallacy when touch sensation is fairly retained, that the patient receives impressions from the skin which help his judgment as to the position of his limbs. Here you see, as in the former tests, there is some uncertainty as to what is the correct reply to the questions asked.

So that in this patient we have found that there is a certain amount of obstruction to the various sensory paths, that he does not feel distinctly on several parts of his limbs, that in places he has no sensation of pain, that in many parts he is unable to say what is the difference of temperature,— whether hot or cold—and that several times he was quite uncertain as to which way his legs were moved and where they were, so that there are sufficient results to illustrate what we have been talking about, viz.: the different paths for the different sensations, and also the exaggeration of reflexes which goes on when there is a lesion in the cord above the lumbar enlargement.

A CASE OF PLEURITIC EFFUSION TREATED BY PARACENTESIS AND ICE

This patient came in on the 29th September. complaining that a week previously he had been seized with pain in his right side, and over the upper part of the right abdomen, and that since then he had grown short of breath. Now, when you hear that combination of symptoms you always suspect that the patient has pleurisy; shortness of breath coming on with pain in the side, and especially pain felt at the terminations of the intercostal nerves, affords ground for a strong suspicion of pleurisy, and when we came to examine this patient's lungs we found that there was dulness over the whole of the lower part of the right chest, loss of breath sounds, of voice sounds, and of vocal fremitus. His heart's apex beat was a finger's breadth outside of the left nipple line, and there was no cardiac murmur; the heart was simply displaced to the left. Thus you see that we had all the characteristic symptoms of an effusion into the right thorax. That being so, we at once aspirated, and with the aspirator removed a pint and a half of serum. Looking at his temperature chart it would have been impossible to say whether the fluid was pus or serum. His temperature in the morning was generally 99° and in the evening rose to 101°, which might well have been indicative of pus, but there was nothing but serum. When we had drawn off the pint and a half of serum from his chest, we did what I think is very useful in such cases, we applied an ice-bag over the affected portion of the chest with a view to diminish the tendency to a reaccumulation of fluid. He kept the bag on for eight days, and did not object to its presence, and the result has been that the heart, which directly after the operation returned to its normal position, is still in its normal place, and when I percuss the affected side you hear that practically there is very good resonance, except over a small area in the posterior axillary region. If I listen I find the breath sounds still somewhat feeble, but the vocal fremitus is very well marked. There can be little or no fluid in the pleura now, so that the ice has been successful, as I hoped it would be, in checking a fresh effusion of inflammatory fluid. We see therefore, that the results of the application of the ice-bag seem to have been immediate and exceedingly good. It is a method which I have frequently used, with advantage, in cases where the pleura contains simply serum. The fluid is at

once drawn off, and then the keeping of an ice-bag on the affected side for some days always tends to hinder the fresh secretion of fluid.

There is another point to which I would draw your attention, and that is the advantage of making the patient take deep draughts of air into the lungs after the fluid has been removed. The lower lobe of the lung has been compressed for some time by the inflammatory fluid, and it takes a little time for it to get into full working order, and the patient can help a great deal in that by taking one or two forcible inspirations and then holding his breath : this expands the lungs and it is followed by deep inspirations which are also of service. And if I percuss his right base while he is thus holding a deep inspiration, you notice at once that the note is distinctly improved.

A CARDIAC CASE.

Here is a case of cardiac disease of considerable complexity, but if one or two of you will examine it under my supervision, I think I shall be able to show you how much you may learn from careful examination of the heart apart from auscultation. Students are far too prone to rely solely on the use of the stethoscope, and to omit altogether the other methods of examination, and yet these yield information which is really more valuable than that obtained by auscultation. Never neglect to acquire by inspection, palpation, and percussion, a correct estimate of the size and strength of the left and right sides of the heart respectively, to note any indications of former pericarditis, and to feel carefully the pulse and the tension in the radial artery, before you proceed to apply your stethoscope. Let us apply this method in the case of this young man of 19 years of age, and when we have made out what we can, I will tell you his history.

First, you inspect the precordial region, and what do you note ?

Two small scars on the skin, which you recognize at once as the result of leech-bites, some distinct prominence of the precordial region showing decided pulsation over a considerable area, and some suspicion of systolic retraction in the intercostal spaces. What may we infer from these observations ? The leech-scars almost certainly indicate previous pericarditis, and the systolic retraction suggests the existence of pericardial adhesions, probably with some pleural adhesions also. The precordial prominence and the obvious pulsa-

tion suggest enlargement and hypertrophy of the heart.

Next, employ percussion; and that not aimlessly, but with the intention of definitely ascertaining the size of the heart by defining its limits in all directions. And bear in mind two points, first, that the left limit of dulness by no means necessarily coincides with the position of the impulse, and, secondly, that the limit to the right is quite as important as either of the others, and is usually as easy to determine. In this case we find the upper limit at the second costal cartilage near the sternum, the left limit at $2\frac{1}{2}$ fingers' breadths outside the nipple line, and the right limit two fingers' breadths to the right of the sternum. I reckon in fingers' breadths, and not in inches ; the latter is apparently more scientific, but is practically not more accurate, and whereas you may not have an inch measure available, you always have fingers. And you will find that two fingers' breadths are almost exactly equal to an inch and a half. What then have we learned from our percussion ? Clearly that the heart is much enlarged, and we know that this may result from pericarditis, or from valvular disease, or from other causes.

Next, let us apply palpation. We observe, first, that over the position of the right ventricle there is a strong heaving impulse, indicating that it is much hypertrophied. But we notice also that outside the left nipple-line the cardiac action is comparatively feeble, and that towards the left limit of the dulness there is hardly any impulse to be felt. This suggests to us that the left ventricle is much less hypertrophied than the right, and we at once feel sure that the latter is doing its best to overcome an obstruction between the two, and we suspect the existence of mitral stenosis. This suspicion is made a certainty when we detect just outside the nipple-line a short but distinct presystolic thrill.

We now examine the pulse. We find that though not frequent, it is small and slightly irregular, qualities probably due to the mitral disease. But at the same time we notice that the wave is sudden, and quite short, features which make us almost certain that there must be some aortic regurgitation as well as mitral stenosis.

Lastly, we find that the veins of the neck are not distended or pulsating ; that there is no enlargement of the liver, and no dropsy. From these facts we infer that the cardiac lesions are for the

present fairly well compensated by the vigorous action of the hypertrophied right ventricle.

Without using the stethoscope at all we have arrived at the conclusion that this patient certainly has mitral stenosis, that he has almost certainly some adhesion of the pericardium, and that he probably has some aortic regurgitation, that the former valve lesion is the more important of the two, and that it is at present well compensated.

And now what does the stethoscope teach us? It reveals a presystolic murmur in the nipple line and a faint diastolic murmur in the second right space, confirming our diagnosis of mitral obstruction and aortic regurgitation. It tells us that the pulmonary second sound is loud, indicating that the tension in the lungs is well maintained by the right ventricle, which we had already inferred.

The only additional facts which it reveals, are that there is a feeble systolic murmur at the left apex, and a much louder and harsher one over the right ventricle, indicating that there is some regurgitation through both auriculo-ventricular orifices. But we must not be misled by the loudness and roughness of the tricuspid murmur into thinking that there is considerable regurgitation through this orifice, for we have already seen that there are no signs of back-pressure. In cases where there is evidence of much back-pressure, you often find no tricuspid murmur at all. So that the stethoscope may actually mislead you, unless you safeguard its results by observations made in other ways.

The history of this young man is as follows: He was first in this hospital when ten years old, and during the following nine years has been an in-patient seven or eight times. He suffered from mitral stenosis, and from many of those attacks of paroxysmal dyspnœa common in this condition, and due, as I believe, to temporary failure of the right ventricle, and a consequent reflex to the respiratory centre. Such attacks are accompanied with some pain, and in him this has usually been the first symptom, but it is not so distressing as the dyspnœa. Pallor of the face and limbs, and often sweating, are features of the attack, which may last, if unchecked, for several hours. Fortunately, rapid relief may be given by the subcutaneous injection of four minims of liq. atropinæ, or by a smaller quantity with a little morphine added. He has often had injections of this kind, and he says he cannot remember that they ever failed to relieve him. During the last year or two these attacks have been less frequent. Only one

has occurred lately; it was relieved by the inhalation of amyl nitrite.

Last January he was again admitted, but this time with pericarditis. Two leeches were at once applied, and then an ice-bag was placed over the heart. This gave him great relief, and the pericardial rub, loud on admission, soon became inaudible. When the attack was over, it did not seem to us that the cardiac dulness was any larger than before. No doubt his aortic valves were attacked at the same time, for since then signs of aortic regurgitation have appeared.

A CLINICAL LECTURE

ON

FACIAL ERYSIPELAS.

Delivered at St. George's Hospital, Oct. 22, 1894, by
JOHN CAVAFY, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—I propose to ask your attention to-day to some of the characteristics of facial erysipelas, of which we have just had an illustrative case, now convalescent, in William King Ward. The patient is a baker, aged 38, and was admitted under my care on October 3rd—on the fifth day of his illness. This, he told us, began by redness and painful swelling of the skin of the right side of the nose, near the nostril, and in immediate contiguity with a small excoriation, which he had scratched. The swelling and redness gradually spread over the right side of the face, and upwards to the lower eyelid. There had been no rigor, delirium, or other sign of severe constitutional disturbance, and he had had no previous attack. The family history showed a strong predisposition to acute rheumatism, from which he said all his brothers and sisters had suffered, and he himself three times; the first attack having occurred when he was 12 years old, and the last, two years and a half ago. On admission, we found the parts above mentioned of a dusky-red colour, hot and tender to the touch, and slightly œdematous; the redness and swelling were sharply circumscribed, especially at the lower and outer part of the cheek, where there was a hard, raised margin. I failed to detect any abnormality of the heart, though others thought the first sound at the apex "murmurish," a sign of somewhat indefinite value.

His tongue was furred, his pulse 116, soft and regular, and his temperature 100.4°, urine high-coloured, acid, free from albumen. In the evening his temperature rose to 102°, with extension of redness and swelling to the right side of the neck, and both eyelids, which were now swollen and closed. During the next three days there was no extension, and the temperature gradually fell, until on October 6th (eighth day), it was normal in the morning. But in the evening he complained of severe pain in the right eye, and the temperature rose to 102.4°, and on the following morning 103°; severe conjunctivitis was found, the membrane being of a vivid scarlet colour, and there was much muco-pus oozing from the nearly closed eyelids. This steadily and rapidly improved under a boric acid lotion, and on October 8th (tenth day) the morning temperature was again normal, but rising in the evening to 101.4°, with extension to the right ear and right side of scalp, which was tender. This was followed by a short quiescent period, but on October 10th there was an evening rise to 103°, and the inflammation, which seemed to have travelled round the scalp, now showed itself on the left ear and left side of face. From this time there was subsidence, with an oscillation due to slight left conjunctivitis, but on the 15th day of the illness the temperature fell to subnormal, and convalescence was established. The swelling and redness steadily subsided, and were followed by slight branny desquamation, the heart sounds were normal, and the urine remained free from albumen.

The points of interest in this case are, first, the mode of onset, without rigor, which is commonly present in severe cases, especially first attacks. Then the clear history of a slight excoriation, as the starting-point of the affection; this is often wanting in cases of facial erysipelas, although it is more than probable that all erysipelas is really traumatic, the original breach of surface being often so trivial as not to attract attention. Another point is the absence of albumen from the urine; it is very often present in erysipelas, but usually transitory, a subsequent nephritis being uncommon. Then delirium, which is very frequent when the scalp is affected, was entirely wanting in this case; when it is present it must by no means be taken as evidence of meningitis, which is, fortunately, a rare complication. With regard to the skin, there are great variations in the extent and severity of the inflammation, the present case being

free from vesication or bullæ, and of moderate intensity, being especially characterized by the great rapidity of subsidence and onset, the former being shown by the fact that a normal temperature was reached three times before convalescence was established, and the latter by the sudden rises associated with extension, this taking place also to the conjunctiva without bad results. When erysipelas extends, as it may do, to the pharynx and larynx, there may be danger from œdema of the glottis. The characteristic spreading at the margin was well shown in this case, while the extension to the neck and very rapid invasion of fresh tissue are indicative of the form of erysipelas which used formerly to be called *e. migrans*, and which I have seen extend gradually over the whole of the trunk. The fever was most irregular and closely related to the extension of inflammation; but there is great variability in the pyrexia which accompanies erysipelas. Some cases, the majority I think, show a rapid rise at the beginning of the attack, which is maintained throughout with only slight morning remissions, and ends by a rapid fall or crisis; but cases with fever of strongly marked remittent type, with a difference of several degrees between the morning and evening temperatures, are occasionally met with, and in some very mild cases, with only slight inflammation of the skin, the fever may be barely perceptible, or even completely absent. I called attention to the latter form some years ago,* and remembering the occasional apyretic course of other inflammations, and the fact that inoculation of the virus of erysipelas now and then produces in animals a spreading erythema without fever, I concluded that such cases were to be looked upon as true erysipelas of an extremely mild type, and am still of this opinion. But the majority of authors seem to look upon high fever as an indispensable symptom, and would therefore not admit the above cases as erysipelas. I am, however, glad to say that this view is not universal, and that Eichhorst and Sorel both declare that erysipelas may run its course without fever.

There can be no doubt that erysipelas is contagious, but not, I think, very actively so in the ordinary facial cases which we admit to the medical wards; as you know, we do not isolate them, and yet it is most unusual to find the disease communicated to other patients in the same ward, while I cannot call to mind a single instance of its having

* Med. Soc. Trans., vol. vii.

been contracted by house-physicians or nurses. Probably a strong predisposition is a necessary antecedent, by far the most important predisposing causes being the puerperal state, and any wound or even slight breach of surface.   This shows the necessity for caution in those who are attending midwifery cases, or called upon to perform even such minor surgical operations as opening an abscess, or vaccination, if they have recently been in contact with erysipelas.

The diagnosis of facial erysipelas is usually easy enough, but mistakes are not infrequently made by the unwary, who are apt to consider any inflammatory and œdematous condition of the face, especially if the swelling leads to closure of the eyelids, as erysipelatous.   In this way 1 have seen the common " swelled face " of toothache taken for erysipelas, a mistake that is easily rectified. Urticaria, again, and some forms of erythema, attacking the face and eyelids, may simulate erysipelas ;   here the multiplicity of the lesions, their rapid subsidence, and the sudden appearance of fresh ones at distant points, soon clear up the case.   Herpes zoster, in the course of the first division of the fifth nerve, may be associated with so much redness and swelling as to resemble erysipelas ; the fixity of the affection, its strict limitation by the middle line, the characteristic clustering of the flat vesicles, and the dark colour of the scabs to which they give rise, are sufficiently distinctive characters.   An acute attack of eczema of the face leads to such considerable redness and swelling, that the likeness to erysipelas may be often a close one ; but here, again, a little observation is all that is required to elucidate the case.   Eczema is far more likely to attack both sides from the beginning, its lesions may be multiple, its border is ill-defined, and beyond it may be seen an advanced guard of small red papules scattered irregularly ; the surface also is not so tense as in erysipelas.   In the great majority of cases erysipelas is accompanied by considerable constitutional disturbance and fever, from which the other affections I have mentioned are usually free.   Urticaria may, however, be febrile, while erysipelas may now and then, in my opinion, be free from fever.   I think that the chief characteristic of erysipelas is its tendency to invade fresh tissue by *rapid spreading at the margin ;* this we certainly do not find in any other superficially similar affection, and if we bear it in mind, we are not likely, I think, to make many mistakes in diagnosis.

THERAPEUTICAL NOTE.

Thioform.—Salt of bismuth, containing bismuth, sodium, and salicylic acid. Yellowish-grey powder ; light, voluminous, inodorous, insipid ; insoluble in water, alcohol, and ether.   Antiseptic action doubtless due to concentrated solution of dithion formed in secretion of wounds after its application ; this solution of dithion (20 per cent.) kills spores of anthrax in forty-five minutes.   Rapidly dries wound, covering it with impermeable layer, and preventing invasion of pathogenic organisms from without. Especially indicated in burns, in which cases it acts with more rapidity and certainty than any other drug.   Dressings should be changed every three days.   Absolutely non-toxic, even ʒiiss at one time causing no bad effects.

(*Nouveaux Remèdes.*)

LETTER TO THE EDITOR.

To the Editor of " THE CLINICAL JOURNAL ":

SIR,—In Sir William MacCormac's lecture on "Stricture of the Urethra," published in your journal of the 25th July, 1894, he is reported to have said in regard to the late Mr. Syme, "He himself, it is said by wicked persons, used to have his patients first examined in the ante-room outside the operating theatre, where, before bringing them into the theatre, a long time was sometimes spent in attempts (which occasionally failed), to pass an instrument through the stricture, and only when this was successfully accomplished was the patient brought in and the subsequent steps of the operation completed. This may be gossip, but I have heard it spoken."

If by "gossip" Sir William MacCormac means a fabrication he is correct.   Though he may "have heard it spoken," he is not thereby justified in repeating and publishing such a statement against an eminent surgeon who is not alive and able to defend himself.

Like many other falsehoods the above misstatement has some foundation.   The late Mr. Syme used to see his patients in a little side room.   Many of these patients were stricture cases, and came from time to time to have bougies passed.   That Mr. Syme carefully tested his cases in private before producing them before the students is not true.   No surgeon was ever more fearless of what anyone might think of his performances in public, or more successful in passing bougies in cases of tight (or so-called impermeable) stricture.   There are many of Mr. Syme's pupils alive and able to testify to the truth of what I have stated.—I am, Sir, yours, etc.,

A. G. MILLER.

EDINBURGH, 30*th July*, 1894.

THE CLINICAL JOURNAL.

TWO CLINICAL LECTURES

ON

FIBROID DISEASES OF THE LUNG.

Delivered at the London Hospital, July, 1894,

By PERCY KIDD,

M.A., M.D. Oxon., F.R.C.P. Lond.,

Assistant-Physician to the Hospital.

I.

GENTLEMEN,—The subject of to-day's lecture is "Fibroid Diseases of the Lung." By this we mean a transformation of their normal spongy alveolar structure into solid connective tissue. The amount of this change varies very much in different cases. At times there are mere patches of fibrous tissue in the lung, in which case the result is not very serious to the patient. Of these cases I shall not speak to-day. But where a large portion of the lung is converted into fibrous tissue there is a loss of so much aërating surface, and serious general effects on the body ensue.

Let me just remind you of some points concerning the connective tissue framework of the lung. The lung, like nearly every other organ, has a special connective tissue of its own. Of this three divisions are commonly made: (1) The subpleural, (2) the interlobular, (3) the peribronchial connective tissue, including under that head the perivascular, as the bronchial tubes and large vessels, running close together, have a more or less common sheath investing them. Remember, too, that the whole of the lung is in continuity in virtue of its lymphatic system. There is, therefore, an easy means of passage from one part of the fibrous tissue of the lung to another part; and we shall see that, inasmuch as fibrous affections of the lungs are of a chronic inflammatory nature, a similar change is very easily produced at some distance from the original focus of disease.

This fibroid change in the lung, as in other organs, may be stationary, or it may be progressive. In the stationary form it is mostly confined to small points, and is not very important;

but when it is progressive the result is very much more serious.

In considering fibroid diseases of the lung, as in the subject of pleurisy, of which we spoke the other day, the question of classification naturally comes up. And now, as then, I should like to say that the most important classification by far is the ætiological. We may divide them, roughly speaking, into two groups—tubercular and non-tubercular. Of these groups the first is unquestionably the more important, because under it are included by far the larger number of cases of fibrous induration of the lung. Tubercle, in other words, is infinitely the most common cause of induration of the lung. But there are several other causes which we shall consider in the next lecture. To-day I want to discuss the subject of fibroid induration of the lung in connection with tuberculosis only—"tuberculous cirrhosis" you may call it. You might ask why we do not call it fibroid phthisis. There are various reasons against the use of that term. In the first place it has been used in different senses. Some would restrict it to a non-tubercular induration of the lung; others would use it for the fibrous forms of tuberculosis; and I think Sir Andrew Clark himself must have had some misgivings about the term, because in one of his last lectures he spoke about "bacillary" and "non-bacillary" phthisis after heading his lecture "fibroid phthisis." I venture to think that this is not an advantage, because it simply introduces fresh confusion into a subject already sufficiently confused. Another objection to the use of the term "fibroid phthisis" is that in every case of chronic induration of the lung there is always a certain amount of fibrosis. And yet for clinical purposes we want to distinguish between ordinary cases of chronic tuberculosis with limited induration, and those cases in which the fibroid element predominates. We shall speak in this lecture of the latter form. For some years I have ceased to employ the term "phthisis" altogether in pathological work, in view of the confusion that obtains in connection with that ancient name, although it was long before I could accustom myself to dispense with its use in clinical medicine.

It should be remembered that from the present standpoint phthisis is a tubercular disease, *i.e.*, a chronic infective disease. Pulmonary tuberculosis may be divided, like other diseases, into an acute and a chronic form. In the acute form we have a predominance of the necrotic or destructive element, whereas in the chronic form fibrosis plays an important part, and may even be the prevailing feature. As a distinguished French pathologist well said several years ago, before the discovery of the tubercle bacillus, "tubercle is a fibro-caseous neoplasm"; and that really puts the whole thing in a nut-shell from a pathological point of view.

The cause of the difference in type which the disease presents in its victims is still a mystery to us. It may be due to certain conditions affecting the individual himself—affecting, that is to say, the host of the parasite; or it may be due to conditions affecting the germ or microbe. The probability is that it depends mainly on the varying degrees of resistance manifested by the patients; in other words, that it is a question of soil.

I shall say nothing of the acute or necrotic type of pulmonary tuberculosis, but shall confine my remarks to the fibrous, indurative form.

The localization of tubercular cirrhosis is a most important matter. Tubercular disease of the lung, with very few exceptions, commences at the apex of the upper lobe. I here show you a specimen illustrating this—a lung very much shrunken and riddled with small cavities and enlarged bronchial tubes. In the earliest stages the fibrous growth commences as a nodule. If you look at the sections I have passed round you will see that the lung tissue contains a number of little black nodules, and if you examine them you will see that they consist of little more than connective tissue, somewhat concentrically arranged with a considerable amount of carbonaceous pigment in them. These are old fibrous or fibroid tubercles. The section is taken from a patient with "knife-grinder's phthisis," a very chronic form of phthisis; indeed, until recent times it was considered doubtful whether it was tuberculous or not. This case occurred in the early days of the tubercle bacillus. One of my colleagues maintained that it was not tuberculous even at the post-mortem examination; and it was not till I had made sections and exhibited the bacillus in some parts of them that I was able to convince him. In a later stage, as seen in a second section, the nodules become fused together. In the first instance, the nodules are

discrete; in the second section, they are clustered and confluent. If you look at the third section you will see that it consists of an almost homogeneous connective tissue. But at its upper edge you will see a minute cavity, a small caseous remnant softening at its centre.

The indurative tubercular process, as in other forms of pulmonary fibrosis, travels along preformed lines. There is thickening around the bronchial tubes, between the lobes, between the lobules, and in the alveolar walls, extending sometimes up to the pleura. To discuss fully the question of the histological changes in tuberculosis would take me too far. I shall pass them over, and merely tell you that this probably begins with a specific bronchitis, leading to a localized broncho-pneumonia. Induration of the connective tissue in the peribronchial sheath ensues, and extends thence to the interlobular and other connective tissue tracts of the lung. Thickening and adhesion of the pleura are always present in this disease.

In tubercular cirrhosis we note one or two differences from the non-tubercular form. First of all, there is the locality—the apex of the upper lobe. Then we very commonly find in the fibrous tissue, or in its neighbourhood, evidence of a definite tuberculous process, caseous nodules, or miliary tubercles, whether singly or in groups, which stamp the whole process as tubercular. In the midst of this fibrous tissue, moreover, you very commonly find excavation. The formation of vomicæ is not absolutely characteristic of tubercular cirrhosis, as we shall see, because we find the same sort of thing in non-tubercular cirrhosis. But the co-existence of tuberculous lesions enables us to recognize the true nature of such cases on post-mortem examination. A conspicuous change in all chronic structural diseases of the lung is pigmentation. In the first section I showed you, marked pigmentation is found, as in the specimens I now pass round, one of which represents a non-tubercular cirrhosis of the lung. There is every reason to believe that all this pigmentation in both cases is really derived from the atmosphere, consisting of inhaled carbonaceous matter which has been absorbed by the bronchial and alveolar lymphatics, and so become diffused through the lung.

In tubercular and in all chronic diseases of the lungs where cavities form we find a liability to the development of aneurysms of branches of the

pulmonary artery. This is thought to be commoner in tubercular cirrhosis, but precisely the same thing may occur in non-tubercular cirrhosis. Here is a large aneurysm in a cavity of the lung from a case of chronic tuberculosis. The existence of such an aneurysm, you can understand, is a very serious danger to the patient; in fact, the rupture of these aneurysms is one of the recognized methods of termination, in chronic forms of pulmonary tuberculosis.

A word or two about the method of extension of tubercular disease, because, as we shall presently see, this has a very important bearing on physical diagnosis. I must be satisfied with giving you a brief statement of fact, without entering into much explanation. The tubercular process extends in the lungs in the following way:—First of all, it may extend by direct continuity; but even then, as a rule, the process extends along the lines of least resistance, that is to say, if a tubercular mass invade a lymphatic space, it will spread along that space in preference to forcing its way through any less defined passage. Again, tubercle bacilli may be carried in the lymphatic circulation for some little distance in the lung, and there set up a fresh focus of disease. These are important methods of extension, but a still more important one remains to be described. A cavity once formed in the lung, its secretions escape into the corresponding bronchus, and are expelled by cough. But the discharge of the sputum is apt to be incomplete, and either owing to the fact that during the deep inspiration following cough or to other causes, infective material is liable to be sucked back into other bronchi, and into healthy lung tissue, and thus arise fresh centres of disease. Without going further into this question, I may say that the distribution of the secondary foci of tuberculosis follows the line of the bronchial tubes, and not that of the vessels or lymphatics.

In speaking of the resolution or cure of pulmonary tuberculosis, I must again be satisfied with a brief statement. Taking the simplest case —that of a tubercular nodule in the lung, the disease may become encapsulated, a fibrous ring developing round about it, the caseous tissue may gradually become inspissated or be converted into lime, giving the appearance of a chalky or stony mass surrounded by a fibrous capsule, which may shrivel up and remain in the lung without further change for years, and give no more trouble. The disease may, however, be arrested short of this.

The tubercular process may never go on to the formation of a definite caseous mass, and we may get simply a small, blackish fibrous nodule or tubercle, which may remain perfectly quiescent for the rest of the patient's life.

The question arises whether we may expect healing, once a definite cavity has formed in the lung. That is a hard question to answer, but my impression is that such a cavity probably never closes altogether. It may shrink to some extent, it may perhaps alter very much in its secreting character, possibly the tubercle bacillus may be gradually starved out in the cavity, and the tubercular process thereby arrested to a large extent; but I doubt very much whether a cavity of any size ever becomes obliterated, though some authorities believe that this actually happens.

Before passing on to the clinical aspects of the disease let me briefly bring together the distinguishing marks of tubercular cirrhosis. There is first the localization,—the apices of the upper lobes are the parts first affected. We frequently find, together with fibrous changes in the lung, caseous nodules, or actual tubercles. There is a great tendency to the formation of vomicæ. The destruction of the lung, as a rule, predominates over the formation of fibrous tissue. There is shrinkage of the cavities with comparatively little fibrous tissue between them. The other lung is very commonly affected with some degree of tuberculosis, often a very extreme degree, as time goes on. And lastly, in other organs of the body also, we may find evidences of the tubercular nature of the disease,—caseous, bronchial, or mesenteric glands, for instance, being present, or perhaps, some old tubercular ulcer in the intestine, or some other indication of the original disease. In chronic forms of pulmonary tuberculosis we commonly find considerable distension of the lung in the vicinity of the fibrous part. When one lung is contracted the other becomes very much enlarged. This enlargement of the lung, which is sometimes called emphysema, is not necessarily a true emphysema, that is to say, a structural change in the lung, leading to rupture of the alveolar walls, and to the formation of bullæ. It is more correctly described as an actual hypertrophy of the lung, in which individual alveoli become enlarged. This is a conservative process calculated to counterbalance the great diminution of the aërating capacity of the lung in the contracted part, by the development of

an increased alveolar surface in neighbouring parts or in the opposite lung.

One more point, and I have done with the pathological part of the subject. Bronchial dilatation is a common occurrence in the more chronic forms of tuberculosis, as in all fibroid affections of the lung, though it is not an invariable change. I shall not go into the pathology of bronchiectasis on the present occasion.

We must turn now to the clinical side of the question. And, first of all, let us take the question of heredity. In a certain number of patients with this form of pulmonary disease we find a history of tuberculosis; but hereditary predisposition is less common in these cases, perhaps, than in the ordinary forms of tuberculosis. Whether it be present or not, I think you should attach very little importance to it from a diagnostic point of view. A history of heredity is important with regard to the prognosis, but not of much value with regard to diagnosis. We must diagnose the disease in the individual, and not be biassed by the question of heredity.

The age of the patient is of some importance. As a rule, tuberculosis manifests a greater chronicity in old patients than in younger people, though exceptions are to be found to this as to every rule. The history of these cases is nearly always a very chronic one, resembling that of chronic bronchitis. The patient has had a chronic cough for years, losing it in summer, but manifesting it in an aggravated form with the return of winter. In other instances the patients have had very little cough at all, although on examination you may detect marked disease of the lung. On being pressed for information, however, the patient will often tell you he has a cough that does not trouble him much, and which he describes as a " constitutional cough." Another patient may come for advice on account of constantly recurring hæmoptysis, and may tell you he was quite well until one day he was seized with a fit of coughing, during which he brought up florid blood. Such patients often have no cough at all, and but for the recurrence of hæmoptysis now and again they feel perfectly well. In some of these cases examination will disclose pulmonary disease, but in others physical signs may be absent or equivocal. Many a person whose chest had been previously examined with a negative result has shortly afterwards died of violent hæmoptysis, and at the post-mortem examination a ruptured pul-

monary aneurysm and old fibroid disease have been discovered. Remember, then, that a hæmoptoic onset is by no means uncommon in this chronic form of pulmonary tuberculosis. But the typical history is that of chronic bronchitis. There is a varying amount of cough, which at times is paroxysmal. The patient will state that he is fairly comfortable all day, but that he has a violent coughing fit in the morning, and that until he has brought up some tenacious mucopurulent expectoration he is very uneasy and short of breath. Once that is expelled he feels well for the rest of the day. Certain patients from time to time expectorate little bits of chalky matter. A patient with this form of disease, every three or four months, used to bring me a paper envelope containing five or six pulmonary calculi—some as big as half a pea. No doubt these came from small cavities in his lung. In fact, the old writers used to speak of a special form of phthisis under the name of "phthisis calculosa."

The expectoration varies very much. It is generally mucopurulent, like that of chronic bronchitis. Dyspnœa, as a rule, is not very much complained of. The patient will tell you that, unless he does anything unusual in the way of exertion, he is not troubled with his breath. He will sometimes complain of palpitation, and you may, at times, find the reason for that in some enlargement of the right side of the heart. The pulse in such cases, of course, is likely to be rather feeble.

A very important symptom—you cannot call it a constant symptom—is hæmoptysis. Hæmoptysis, when it occurs frequently, affects the prospects of the patient very seriously. Such persons are liable to succumb from the direct result of the hæmorrhage. The method of death in these cases of profuse hæmoptysis is mostly suffocative; it is not due to the amount of blood lost, but to the blood poured out from the ruptured artery escaping into the bronchial tubes. In the violent inspiratory efforts which ensue blood is sucked back into the terminal bronchi in sound parts of the lung, producing an obstruction of the bronchial tubes like that which occurs in death from drowning, so that these patients die from a form of asphyxia. At the post-mortem examination you find hæmorrhagic patches in the lungs, representing lobules stuffed full of blood.

Pyrexia is, for the most part, absent in a typical case of this disease, though the patients are liable

to spells of fever from time to time. It is well to be on the watch for this, because the supervention of fever in a case of chronic tubercular cirrhosis may perhaps tell you much more than the physical examination of the chest. Thus on making a fresh examination of a patient, whom you had examined a few weeks before, you may find no definite difference in the physical signs, although the patient is obviously worse, his temperature rising at night to 101° or 102°, and falling in the morning to normal or 99°. From the presence of pyrexia in these cases you may conclude that, although the case is chronic, the disease is, to some extent, progressive. But cases of this sort may go on for months, or even for years, with no noteworthy pyrexia at any time, differing in that respect in a very important way from the ordinary forms of pulmonary tuberculosis in which pyrexia is so very common a symptom.

Wasting of the body, in the sense of progressive emaciation, is not, as a rule, met with in these cases. A patient who has had this disease for some years is generally thinner than an average person, but you must not attach too much importance to that. You want first to know the bodily habit of your patients. Some are naturally thin. The prognosis will depend very much on the history—whether the patient has lost flesh recently, and is still losing flesh. If there be progressive wasting, it has, of course, very much the same significance as pyrexia, that is, it indicates progressive disease.

Night sweats, too, are uncommon, unless there is active disease, and they are mostly associated with pyrexia, and other constitutional symptoms.

The digestive system is a very important one in this connection, and should invariably be investigated, because the best results obtained from treatment are obtained when we concentrate our attention on this point. As long as a patient's appetite is good, as long as he is not losing flesh, and has little or no rise of temperature, we may prognosticate a chronic course, in spite of the existence of extensive disease in the lung. In proportion as constitutional symptoms are only slightly pronounced, or otherwise, the case is likely to do well or end in a short time.

A few words about the complications of this disease. Acute bronchitis is from time to time met with in these patients, particularly in wintry and foggy weather. A few foggy days may rapidly cut off a patient, who might otherwise have lived for

years. In other patients, chronic bronchitis is set up, and the end comes slowly in the usual manner of such cases : the right heart enlarges, congestive œdema occurs at the base of the lungs, and death occurs from slow asphyxia.

The development of acute bronchitis, however, may be due not to atmospheric causes, but, as post-mortem examination demonstrates, to an acute eruption of tubercle in the lung. As long as you have a cavity in the lung secreting the specific tubercular poison this is liable to occur, the germs becoming dispersed to other bronchial districts. Occasionally a constitutional extension of the disease is observed, the patient dying from acute generalised tuberculosis. General infection is probably the result of tubercular invasion of a small pulmonary vein and the entrance of large numbers of tubercle bacilli into the systemic circulation. This is a rather rare termination of these cases.

As the result of the long-continued suppuration, existing in cases of this sort with secreting cavities, amyloid degeneration is not at all uncommon, and this constitutes another cause of untimely termination. The amyloid disease of the intestine, kidney, liver, or whatever part of the body is affected, leads to profound alteration of the nutrition of the body. The patient becomes very anæmic, suffers from diarrhœa, or may exhibit albuminuria and other symptoms of renal disease. In other instances there is no albuminuria or diarrhœa, but enlargement of the liver or spleen is detected. The simultaneous enlargement of both organs co-existing with a marked degree of anæmia is very significant of lardaceous degeneration. This condition is occasionally observed without any marked history of suppuration, and in these cases you may find the patient has had syphilis—a very important cause of amyloid disease.

Pulmonary aneurysm is an extremely common complication, and is a relatively frequent cause of sudden death.

Tuberculosis of other organs, of sufficient dimensions to give clinical evidence of its presence, is not common. The larynx and intestine are seldom affected, but it is much more common to find tubercular ulcers in the intestine than in the larynx. Tubercular changes in the lymphatic glands are not unfrequently found post mortem; but during life we do not find much evidence clinically of an extensive general tuberculosis.

Pleurisy, in the acute form, is very rare ; pneumothorax is practically unknown.

Before going on to the subject of physical diagnosis, I wish to refer to one more clinical point—namely, the fact that putrid expectoration is very rare. That is an important point in relation to diagnosis, for when we come to speak of non-tubercular cirrhosis we shall find that putrescence of the secretions is very common. Putrid sputum, in a case of contracting lung disease, would justify the opinion that the disease is not tubercular; speaking generally, I believe this to be true, though there may be exceptions. What the explanation is I am not prepared to say; but I shall allude to this again when I speak of bronchiectasis.

In complicated cases, the course of the disease is slowly progressive. The opposite lung, and the other parts of the affected lung, become involved in the tubercular process, and the end of such patients does not differ greatly from that of an ordinary case of phthisis, which runs its course in two, three, or four years. The progress of the case largely depends on the existence of complications, hæmorrhage from the lungs, acute bronchitis, and the development of acute pulmonary tuberculosis and amyloid disease being the most important. The co-existence of morbus cordis (cardiac dilation, the result of valvular disease, or other causes), with pulmonary tuberculosis, almost ensures a chronic course for the disease, and the patient lives much longer than he would if he had pulmonary tuberculosis alone. Emphysema also seems to produce a tendency to chronicity.

We come now to the physical diagnosis. I shall not attempt to go into great detail here, because I hope to have one or two patients at the next lecture on whom to demonstrate the physical signs before you, but I shall give you a brief sketch of what seem to be the most important points.

The physical evidence of fibroid or cirrhotic tuberculosis may be briefly said to depend on the presence of a contractile lesion in the apex of the lung. We find dulness on percussion, a diminution of the movement of the chest, and a flattening or retraction of the chest wall, all pointing to the existence of a fibroid, contractile pulmonary disease. The dulness which you get in these cases is, as a rule, extreme, approaching in degree to that obtained in fluid effusions, and in these cases is often largely due to thickening of the pleura, the thick pleura giving a very dense dull percussion note. Other important indications of the shrinking nature of the disease are afforded by displacement of different organs.

First, let us consider the displacement of the heart. When the upper lobe of the left lung is the seat of a cirrhotic tubercular process, the apex of the heart is displaced outwards, and more or less upwards. The shrinking lung retracts towards the clavicle; the heart is drawn up, and the apex beat is found in the fourth instead of the fifth intercostal space. The impulse of the heart may reach as high as the second rib. Many a time has this condition been diagnosed as dilatation of the heart, from the extended area of cardiac impulse and dulness. But before you even offer to make such a diagnosis, you must be careful to exclude disease of the lung. The contraction causes a recession of the anterior margin of the lung from the surface of the heart, which thus comes into contact with the chest wall, and cardiac pulsation is felt over a large area. There is this difference between the displacement of the heart in tubercular and non-tubercular cirrhosis, that the heart in the tubercular form is more apt to be raised, as well as displaced outwards, due to the fact that the contracting process starts in the apex of the lung. The non-tubercular process starts, generally speaking, in the lower lobe, consequently, the displacement is purely lateral—to one side or the other.

As the result of this displacement and uncovering of the heart, systolic murmurs are often produced at the base, especially on the left side. Such murmurs are probably due to traction of the lung on the aorta or pulmonary artery, producing a slight narrowing of the vessel.

In addition to displacement of the heart, upward dislocation of the diaphragm and abdominal viscera may be produced, the liver on the right side, the stomach or colon on the left, being drawn up into the thorax. On percussion over the lower part of the chest, in cases of contraction of the left lung, a curious tympanitic note is obtained like that found over the colon or cæcum.

Excavation. As a rule, in cases of contractile tubercular disease, we find evidence of excavation—tubular or cavernous breathing, pectoriloquy, bronchopony, or, still more important, various abnormal sounds produced by cough. I shall not go into these at any length, as I may be able to demonstrate them to you on some patients, which will be much more useful to you than any description. One sign I must refer to, because it is a very valuable one. When the patient coughs you hear a hissing expiratory sound, followed im-

mediately by a short sucking inspiration—a sound which Dr. Mitchell Bruce has well described as " the india-rubber ball sound." If you squeeze an india-rubber ball and suddenly let it go, you will get a very good representation of this phenomenon.

In addition to the apical signs of excavation and contraction, we commonly find at other points râles with or without slight dulness and modifications of the breath sounds, pointing to localized bronchitis or consolidation of tubercular origin. There are certain regions which should be specially investigated for evidence of secondary disease. First of all, you should always examine the apex of the opposite lung most carefully. Both lungs should, of course, be thoroughly examined in every case, but I would particularly advise you to examine the supraclavicular and supraspinous regions, and, in addition, the infraspinous fossa which corresponds to the apex of the lower lobe. The interlobar septum, dividing the upper and lower lobes, runs from behind downwards and forwards, being placed at its commencement close to the vertebræ, about the level of the spine of the scapula. If, adopting Dr. Fowler's suggestion, we make the patient put his hand upon the spine of the opposite scapula, while the elbow is raised above the level of the shoulder, the vertebral border of the scapula indicates very closely the line dividing the two lobes of the lung.

In many cases of contracting disease of the upper lobe, you will find the lower lobe quite free from morbid sounds, with the exception of the infraspinous fossa, i.e., the apex of the lower lobe. This is a point of considerable importance, because the tubercular process has a marked tendency to advance along certain lines, these being marked out by the course of the bronchial tubes. A large straight bronchus, as pointed out by Dr. Wm. Ewart long ago, is given off nearly horizontally to the apex of the lower lobe, and the proclivity of this part of the lower lobe to secondary tubercular disease is to be explained by the ease with which infective material from cavities in the lungs can be drawn into the large straight bronchus supplying it.

In estimating the degree of the disease you must not trust exclusively to physical examination. Remember that the compensatory distension or hypertrophy of the lung, which so frequently is developed, may, to a large extent, mask the existence of tubercular lesions, and may lead you to think the disease in the lung affected is more circumscribed than it really is.

In all cases, except where the evidence as to the tubercular nature of the process is very complete, it is well to check our diagnosis by an examination of the sputum, because in some instances of chronic induration of the lung it is hard to say whether it is tubercular or not. But in the sputum we have a most reliable means of diagnosis. Therefore, make yourselves familiar with the methods of examination for the tubercle bacillus.

The prognosis in these cases turns largely on the extent of the disease. It depends also on the presence or absence of persistent bronchitis, because physical signs of bronchitis in a case of phthisis always raise a suspicion that the bronchitis is due to disseminated tubercle. A tendency to bronchitis on slight provocation is a bad omen.

The presence of amyloid disease is a most unfavourable element in the prognosis.

Hæmoptysis, when it recurs frequently, is a very unfavourable symptom, because it raises a suspicion that in a subsequent attack, the patient may succumb from the rupture of an aneurysm. But because a patient has had a severe hæmoptysis once or twice you must not therefore give an unfavourable prognosis, as the hæmoptysis may never be repeated, and the case may run a very chronic course.

Always inquire very carefully into the constitutional symptoms, for they form the most important elements of prognosis. The tendency is to expect too much from physical examination. For, while it can tell us much, it cannot tell us all. It can often help us to recognize the presence of disease, but it also often fails to tell us the extent of the disease. We can generally form a fairly accurate estimate if we compare the physical examination with the constitutional condition of the patient and a review of his different symptoms, but it is always well to speak in a very guarded way as to the extent of the disease. It is surprising how long a patient can remain in tolerable health with very extensive disease of the lungs so long as he has no constitutional symptoms—the most important of which are pyrexia, loss of flesh, and anæmia.

Lastly, a history of family predisposition, indicating a faulty resisting power, makes the prognosis less favourable.

I meant to have said a few words about the treatment. But this is a very large subject, and I cannot now go into it at any length. The object I had in view was to give you some intelligent

idea of the different fibroid diseases of the lungs, and to put before you the principles of diagnosis. I must, on the present occasion, omit all reference to treatment, for if I did attempt to treat the subject at all it would require a whole lecture to itself.

COMMON TUMOURS OF THE OVARY AND BROAD LIGAMENT.

A Demonstration delivered to the London M.B. Class, St. Bartholomew's Hospital, Sept. 19, 1894,

By C. HUBERT ROBERTS, F.R.C.S., M.B., M.R.C.P.,

Demonstrator of Practical Midwifery to St. Bartholomew's Hospital; Physician to Out-Patients, Samaritan Hospital for Women.

GENTLEMEN,—This morning we will consider the question of the causes, pathology, varieties, symptoms, and treatment of the common tumours of the ovary. I have brought you down some diagrams and specimens from our museum, and with them |I will try and make clear to you some of the chief points in the consideration of the various forms of common ovarian tumours. I have written out a schedule on the black board giving the chief headings which we will now consider more fully.

ANATOMY, HISTOLOGY, ETC.

First of all, do not forget your anatomy and histology in this subject. Perhaps you will look at some of these diagrams which Dr. Champneys uses for his lectures, and which he kindly allows me to use for these demonstrations. In this diagram you are looking on the top.
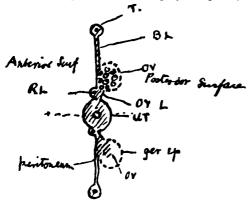
Fig. 1.—Ut. is the uterus, *B. l.* is the broad ligament, showing anterior and posterior layers. In fig. 1 we have the normal condition, and in figs. 2–8 abnormal cases. The continuous line in these figures is the true peritoneum, but the ovary itself is covered with a different epithelium, to which we give the name germinal epithelium (dotted line). It is not exactly peritoneum, but as the ovary was developed in the lumbar region of the embryo, in the region of the kidney, of course it was covered with the original germinal epithelium, and when the ovary descends later into the pelvis it still keeps that original germinal epithelium on its surface, and it is not really true peritoneum. The

value of knowing this is that you cannot strip this epithelium from the ovary, and so when you have a true ovarian tumour you cannot strip the epithelium off. But if the tumour (*e.g.*, broad ligament cyst, figs. 5, 6, 7, 8) grows in between layers of the peritoneum, then you can strip that peritoneum off. That goes to show you roughly when you see a tumour—say, in the post-mortem room—whether it grew from the ovary itself.

In fig. 2 we have the first idea of an ovarian tumour—a multilocular ovarian cyst growing from the glandular portion of the ovary proper.

There is another form in figs. 4, 5, 6, 7 and 8, which are cysts, though not necessarily multilocular, containing papillomatous or warty growths. Those tumours grow nearly invariably from the hilum or broad ligament, not from the glandular part of the ovary itself, though rarely they have been seen in small glandular cysts. In figs. 5, 6, 7 and 8 there are other tumours very well shown, though they are not in a sense ovarian tumours; they do not grow from the ovary. You see the ovary is quite normal, and it is simply displaced with the Fallopian tube. Now, these tumours are broad ligament cysts; whether or not they are parovarian is another matter. As you see in the figures they are quite different. The ovary itself remains covered by the germinal epithelium, but the cysts themselves—the broad ligament cysts—are covered by the true peritoneum. In such cysts you can strip the peritoneum off. It makes a great difference in operations as to whether we get a pedicle, or the cyst has burrowed between the layers of the broad ligament and has to be shelled out.

We now come to another set of diagrams (fig. 10, after Mr. Alban Doran): a diagrammatical section of the uterus with the broad ligaments and structures therein. You have in the broad ligament structures important with regard to the etiology of many of these ovarian new growths. You have, between the layers of the broad ligament, the remains of the meso-nephros and its duct, consisting of straight and vertical tubes. The meso-nephros, the parovarium, and the Wolffian body are identical. Its duct is the duct of Gartner. There are other irregular tubes described by Kobelt, probably the remains of the convoluted tubes of the meso-nephros. In the male there are similar structures—the vas deferens epididymis, vasa aberrantia, etc., in connection with the testicle. With regard to the other portion you see the tube,

Fig. 1.

(Figs. 1–8 copied from Dr. Champneys' Diagrams.)

Trans-section of broad ligaments and uterus, showing relations of peritoneum, ovaries, tubes, etc.

T.—Fallopian tube. UT.—Uterus.
B. L.—Broad ligament. OV. L.—Ovarian ligament.
R. L.—Round ligament.
Continuous line—Peritoneum proper.
Dotted line—Germinal epithelium.
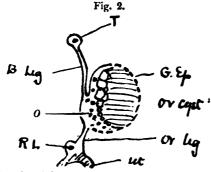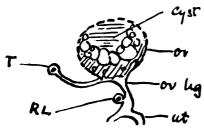
Fig. 2.
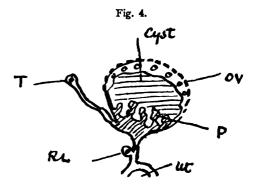
Origin of multilocular glandular cyst, showing remains of ovarian tissue, (o).

Fig. 3.

Showing further enlargement of multilocular glandular ovarian cyst. The Fallopian tube is displaced.

Fig. 4.

Showing origin of papillomatous cyst of hilum, glandular part of ovary displaced to surface of cyst.

P.—Papillomata.

Fig. 5.
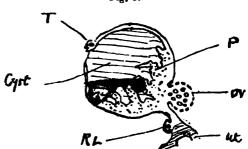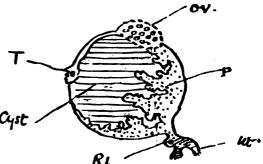
Diagram of papillomatous cyst of broad ligament, ovary normal, tube displaced to surface of cyst.

Fig. 6.

Papillomatous cyst of broad ligament, displacing ovary and tube.

the ligament of the ovary, and broad ligament. You will see that I have put in (fig. 10) the possible places where cysts may arise (dark spaces). I give you a classification—not a very good one, but a fair one—of the cysts. Now, there are tumours which arise from the ovary, and tumours which arise in the region of the ovary; but you would not call them all true ovarian tumours, though when they grow to a great size the primary relations are lost.

CLASSIFICATION OF OVARIAN TUMOURS.

A. *Ovarian Tumours Proper.*
　　　Covered with germinal epithelium, and arising from ovary itself.
　　1. Dropsy of Graafian follicle.
　　2. Multilocular ovarian cysts.
　　　　a. Glandular (adenomata), growing from gland tissue.
　　　　b. Papillomatous, growing from hilum.
　　3. Dermoids.
　　4. Solid ovarian tumours (rare).
　　　　Myoma, fibroma, carcinoma, sarcoma.
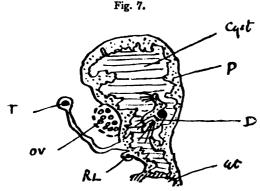
B. *So-called Ovarian Tumours.*
　　　Growing in region of ovary, not primarily arising from ovary proper, covered by peritoneum proper.
　　1. Broad ligament cysts proper.
　　2. Parovarian cysts.
　　3. Papilloma of Fallopian tube or broad ligament.
　　4. Tubo-ovarian cysts.

Now, let us take tumours which grow from the ovary proper (*vide* Classification). First of all,

Fig. 7.
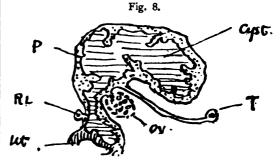
Papillomatous broad ligament cyst, burrowing into layers of broad ligament, having no pedicle therefore; ovary and tube displaced.
D.—Portion of dermoid tissue in cyst wall.

there is *dropsy of the follicle,* which I only specify. But we will pass to the ordinary ovarian tumours: figs. 2 and 3 are very common indeed: you have the *glandular,* multilocular, or adenomatous tumour (adenomatous is a very good name); and the tumour which is *papillomatous* (figs. 5, 6, 7 and 8), according to where they arise. In figs. 2 and 3 you see the glandular tumour arising; and in figs. 5, 6, 7 and 8, there is the papillomatous tumour. They are easily distinguished at once by the growing from the gland part, or growing upon some part of the hilum. It makes a good deal of difference whether you have a tumour growing from the glandular part, or whether it grows from

the hilum: the reason being that the more you get in the region of the hilum, the more you get into the region of the developmental remains, parovarium, etc. Therefore, many of these tumours, at all events, the papillomatous tumours, we presume, grow from the region of the hilum. That is a very important point; only a very few glandular cysts contain papillomatous or warty growths.
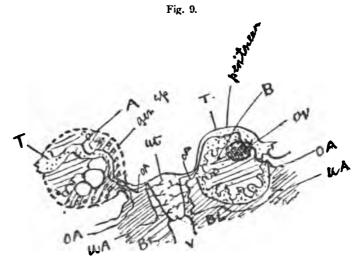
The next class are the dermoid tumours, which are very interesting. I have several here, which you can see afterwards. They are called "dermoid" because they contain structures which are akin to those of the skin. I shall go into that matter possibly later on.

Then we have "*solid ovarian tumours.*" I have a few specimens here, but they are extremely rare. You have myoma, fibroma, sarcoma, and carcinoma. In the next division, or "*so-called ovarian tumours,*" I take *broad ligament cysts.* I have here several specimens of broad ligament cysts,

Fig. 8.

Another broad ligament cyst extending deeply into broad ligament, displacing tube and ovary, papillomatous also.

and they arise either from the parovarium or from some region around, but not necessarily from the tubes of the parovarium, so that all broad ligament cysts are not necessarily parovarian, although a good many of them are. The true *parovarian cyst* (a broad ligament cyst) grows from some of these curious developmental tubes. The *papilloma of Fallopian tube* is an extremely rare condition; and I think that many of the specimens which have been described as papillomata of the Fallopian tube, are really burst papillomatous cysts of broad ligament. Supposing you had a cyst of this nature (specimen shown) full of papillomata, growing from the cyst wall, and that such a tumour burst, it is possible that you could get such papillomatous growth free, or possibly there may be primary papillomatous growths of tube and broad ligament.

Fig. 9.

A.—Glandular multilocular cyst having pedicle.
B.—Papillomatous cyst of broad ligament growing deeply into its layers, ovary not
     affected, tube is displaced, no pedicle.   O.A.—Ovarian artery and branches.
U.A.—Uterine artery and branches.   V.—Vagina.   B.L.—Broad ligament.
Dotted line—Germinal Epithelium.   Continuous line—Peritoneum.

Fig. 10 (after Doran).
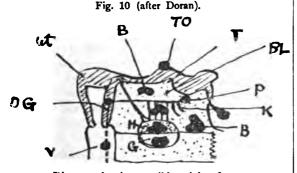
Diagram showing possible origin of cyst.

Ut.—Uterus.   P.—Parovarium.
V.—Vaginal cyst.   D.G.—Duct of Gartner
                          (uterine cyst).
T.—Tube.   H.—Hilum of ovary.
B.L.—Broad ligament.   G.—Glandular portion of ovary.
The cysts are shown arising in the various portions.
T.O.—Tubo-ovarian cyst (?).
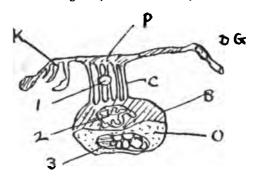
Fig. 11 (after Bland Sutton).

P.—Parovarium.   1—Parovarian cyst.
B.—Paroöphoron.   2—Papillomatous cyst of hilum.
O.—Oöphoron.   3—Glandular cyst (multilocular).
K.—Kobelt's tubes.
C.—Straight tubes of parovarium.
D.G.—Duct of P.—Gartner.

I have seen curious tumours of broad ligaments, with great masses of warty growths, involving broad ligament and tube—they are often bilateral. We have, lastly, a very rare form of cyst indeed. *tubo-ovarian*, which grows, or is supposed by some to grow, from the tube primarily, and affect the ovary simply by contiguity. Possibly some of these tubo-ovarian tumours are really ovarian hydroceles, into which the tube opens secondarily. These are very uncommon, and probably not very important. So much for the classification.

DESCRIPTION OF CYSTS.

It would take a very long time to describe these various tumours. The best way is to see them for yourselves operated upon and after removal, in Martha Ward. We now diagnose these tumours and remove them early, so that one rarely sees women with enormous tumours as one did in the old days. No length of time can elapse without some such tumours being operated on in this hospital.

The common multilocular tumour is a glandular

one. In the specimen I have here, you see, all the multilocular part is inside the great cyst—that is to say, all the cyst formation arises inside the mother cyst. They always grow inside—endogenous, not exogenous—cyst formation. You will notice that the intracystic part in this specimen is practically solid. The tumour is really a cystic adenoma of the ovary. If you take sections of these you will find myriads of tiny distended glandular spaces containing thick sticky fluid; in fact, these cysts are lined with a mucous membrane.

I show you here several other cysts similar to the above.

Now, taking the second variety, not necessarily multilocular, *papillomatous cysts*, this specimen shows very well the papillomatous condition one gets in such cysts growing from the hilum.

Dermoids are very curious tumours. They are small tumours, as a rule, filled with the usual curious things, such as hair and sebaceous greasy matter; cholesterin and secretions, which come from skin or skin structures. Sometimes a dermoid contains more than that. In the walls you will find teeth—generally rudimentary—and sometimes curious knobs and bosses of solid tissue; very remarkable are the masses of tissue like mammary glands in the inside of cyst wall. They were described by Mr. Bland Sutton, in his book, "Diseases of Tubes and Ovaries." Some of the rudimentary mammæ contain gland tissue. Muscle is sometimes seen in dermoids, but it is uncommon. Therefore, we presume that dermoid tumours are *not* essentially epiblastic, but contain also mesoblastic structures.

In this bottle you see the curious yellow, sticky, buttery-looking fluid that may be in such cysts. Again, in this specimen there is a large mass of conglomerated hair which came from the inside of a dermoid tumour. Here is another tumour showing that you can have a tumour partially of one form of tumour, multilocular, and yet in its walls you may have a portion which is dermoid. That, I think, is rather curious.

As I have already said, *solid* ovarian tumours are extremely rare.

Here is a specimen of bilateral solid tumours—two large masses on each side of the uterus; they were malignant. Now, etiologically, since an ovary contains so many compound structures, you ought to, and do get, tumours consisting of these particular tissues. You may have a

myoma, fibroma, sarcoma, and carcinoma but very rarely. It is a sort of axiom that solid ovarian tumours are malignant, but that is not necessarily so. I have no specimens to show you of such tumours. Now look at this big cyst, which, removed by operation, is a very good specimen indeed of a broad ligament cyst, containing papillomata. It contained many pints of fluid, and scattered all over its walls were these growths (figs. 5, 6, 7 and 8) like warts. Indeed, they are identical with warts, and are papillomata. You might say for certain that that cyst grew from the hilum, or broad ligament, and you see I can strip off the peritoneum. Now, these rough prominences which you see are most beautiful things under the microscope. You really get a section very like these adenomatous growths which you get in the rectum. You see the cyst wall, and projecting from it in every possible form are papillæ, covered with one layer of columnar cells, below which are a delicate stroma and blood vessels. The epithelium lining the cyst proper varies with the size of the cyst. I believe, in small cysts, the epithelium is generally columnar, but in big cysts it is flattened out, probably by pressure. These intracystic growths are very curious, because they have the power sometimes, apparently, of reproducing themselves on contiguous structures. Cases have been operated upon where the whole of the peritoneum has been found filled up with this sort of papillomatous, cystic-looking growth, and I believe they have come originally from a burst papillomatous cyst, or from a cyst through which these things have made their way. Very rarely the papillomatous masses may start from the tube or broad ligament without the formation of a cyst. It is not necessary because you find the peritoneum full of these things, to say that they are really malignant. They may be clinically malignant, that is to say, they reproduce themselves, but they are not malignant in the sense of a cancer, and they clear up sometimes after operation. This is why I refer to these tumours in connection with malignant disease of the ovary.

Secondary deposits of melanotic sarcoma I show you here in the ovary. Tubercle also may occur secondarily. Later, I will have microscopes here, and will bring you down sections of ovarian cysts. With regard to *parovarian cysts* I have not very much to say. They are, of course, essentially broad ligament cysts, since they grow between the

layers of the peritoneum, and they are curious in that they are almost invariably unilocular. They differ, too, in the fluid which they contain. Sometimes you will find papillomata in a parovarian cyst. There are other cysts of the broad ligament—that is to say, between the layers of the peritoneum—which are not essentially parovarian. Mr. Bland Sutton in his book describes them in a different way. This diagram is drawn from his book. You see here (fig. 11) the straight tubes and the vertical tubes, forming the parovarium and its duct (Wolffian body), the ovary proper having its two great portions, the glandular part and the hilum. He calls these three portions respectively (*a*) parovarium, (*b*) paroöphoron, (*c*) oöphoron, and from those three divisions all such cysts practically arise. (Compare Doran's diagram.)

Papilloma of Fallopian tube I have dismissed, telling you they were possibly burst papillomatous cysts. The tubo-ovarian cysts I referred to as possibly hydroceles of the ovary. Now I have finished the classification, and described shortly the commoner cysts.

CLINICAL APPEARANCES, ETC.

We will turn now to the appearance of the cyst. You have seen, or ought to have seen, the abdomen opened for an ovarian tumour. What should it look like? As a rule, if it is not inflamed you will find the surface is glistening, whitish or bluish and smooth, and covered with small venules. The next thing that possibly you noticed was that the hand has passed over it to see if there are any adhesions. It is a very important fact that when you come to operate upon an ovarian tumour, if it be inflamed and has adhesions to its surface, the operation may be difficult, especially if the cyst is thin or rotten. Therefore we do not like to see an ovarian tumour which has not a smooth and glistening surface. Sometimes it may be an indication of its contents. Dermoids are often coloured, also inflamed cysts. As the operation continues the cyst is tapped. The tumour is gradually emptied and the pedicle sought for and tied, and the large vessels can frequently be ligatured separately—using thin ligatures—and then the remainder of the pedicle transfixed and tied on both sides.

FLUID.

The common ovarian tumours contain fluid of all sorts. It is generally highly albuminous,

becoming nearly solid when boiled; and besides that it contains a great deal of mucin, and sometimes bodies like boiled sago; its colour greenish, brownish, reddish, yellowish, often due to blood altered pigment. It contains epithelial scales and cells, and may contain crystals of either blood or cholesterin. (Cholesterin in old cysts is common.) The fluid varies with the variety, age and size of the cyst—that is to say, if it is a glandular cyst which grows from the ovary proper you would expect it to contain a good deal of mucin, albumen, and things of that sort; but if it is a papillomatous cyst the fluid is different. It is generally a thinnish watery fluid, containing possibly a little albumen or mucin, but it is not common. The parovarian cyst contains fluid, which is practically salt and water. Of course what a cyst contains varies very much. Look at the dermoid. These specimens which I hand round have greasy, sebaceous, buttery, yellow-looking fluid. Such fluid, of course, is very dangerous. Should any of it get into the peritoneal cavity the results would probably be disastrous. So that the prognosis of the ovarian tumour depends a good deal upon what it contains. You cannot tell what it contains before you operate upon it, and therefore the warning comes early here, "that ovarian cysts are only to be treated by removal," not by tapping. (I do not propose here to go into the etiology of dermoids, which is a very difficult subject.)

DANGERS, COMPLICATIONS, ETC.

Now about the dangers, complications, etc., of the common ovarian cysts. Supposing a patient comes to you with an ovarian cyst. You diagnose the cyst as well as you can; and remember that you ought to diagnose all you can—you want to diagnose everything possible, that is to say, there may be more than one cyst, you may have pregnancy with the cyst, and there may be other complications. When you make your diagnosis, therefore, try and diagnose everything. Now you advise that woman to be operated upon, and the reason is very evident, viz.: that the cyst is very liable to undergo some change. The danger is that you can never tell when that cyst may get into trouble; it may inflame, the pedicle may become rotated, or the cyst may burst. It is not uncommon to see rotation of the pedicle. A woman has an ovarian tumour, we will say; it does not seem to cause her any trouble, but one day by some means or other the pedicle gets twisted, at first the venous and

lastly the arterial blood supply is interfered with, strangulation, and then gangrene of the cyst sets in. Those are very important things. Again, the cyst may burst, and lastly for some reason or other, although we do not get rotation of the pedicle, we may get a cyst which may inflame and suppurate, infected from some other region. The infection of ovarian tumours is very curious; where the sepsis come from that infects them we do not know; but we presume they are infected from the bowels. There must be myriads of micro-organisms in the intestinal cavity. They are not hurtful to us, provided that the resistance of our tissues is not lowered, but when you get such a stage of lowered vitality, then I presume such micro-organisms can affect tissues which before resisted them. It is like an extra-uterine gestation sac. Why does that inflame? Sepsis cannot enter through the uterus; it must get there by contiguity, and I have seen cases where adhesions and the results of inflammation of the cyst have rendered its removal impossible. Then we have inflammation of the contents. Sometimes an ovarian tumour is full of stinking pus. Now, you may conclude that the more dangerous the fluid is, the more dangerous is the tumour should it inflame or burst. Fancy getting the contents of, say a dermoid, into the peritoneal cavity; the result would probably be disastrous, and peritonitis ensue.

The rotation of the pedicle is a very curious thing; why should the pedicle rotate? I do not think anybody knows. There is no particular way, apparently, in which the pedicle is twisted. Sometimes it is twisted one way and sometimes another, and whether it is the movement of the intestinal tract, or the movement of fæcal matter down the intestines, one does not know, but one knows that rotation of the pedicle does occur, and that it is sufficient to stop circulation, and inflammation accordingly takes place. With regard to rupture of the cysts, I may tell you that one does not often see a cyst burst nowadays. We diagnose these cysts and remove them early, and do not leave them to burst, though dermoids, even small, sometimes burst into the adjacent bowel. Bursting of a cyst, except by accident, is, uncommon. Should cysts burst, they either burst into the peritoneal cavity or through adhesions into some contiguous viscus. They generally leak quite slowly and gradually. You seldom get a great rent except by violence. Now, the rupture of such a cyst were it simply parovarian for instance,

containing salt and water, would do no harm; but if it contained dangerous fluid, such as a dermoid, then it would be very serious. I have seen a cyst burst, and one may get a history such as this:—"The patient became suddenly smaller, and the swelling disappeared." Possibly the patient would tell you that she had micturated a great deal. The absorption of the fluid by other vessels, and the passing of a large quantity of urine, together with a bad attack of diarrhœa, would be suggestive that the tumour had burst. I do not say that that is necessarily so, but sometimes that is the case.

Now, apart from the dangers of inflammation, rupture, and rotation of such cysts, they are dangerous sometimes on account of their size. You may occasionally see enormous cysts. They fill up the abdomen, interfere with respiration, and cause severe pressure symptoms. You do not, however, often see them now.

DIAGNOSIS.

The *diagnosis* of ovarian tumours remains with the diagnosis of abdominal tumours. I am afraid that I cannot possibly go over everything that an ovarian tumour might not be. I told you that I thought the best way of attacking the diagnosis of abdominal tumour was by the process of exclusion. I advise you to divide them into two great groups —pelvic tumours and abdominal tumours; and having so done, to make a sub-classification as to whether they were fixed or whether they could be displaced; and lastly, Is it a tumour at all? That is to say, could it not possibly be flatulence, or such things as distended bladder, hydramnios, ascites, encysted or not? You have to make up your mind as to those particulars.

I shall not go into the diagnosis of abdominal tumours here. When you have a case of this sort do not be satisfied with diagnosing one thing, but try and diagnose everything, because it is quite possible you may have other things besides an ovarian tumour or tumours—pregnancy, for instance—which keep always on your mind.

TREATMENT.

Now, lastly, there is the question of *treatment*. There is but one treatment, and that is ovariotomy. Please do not say tapping, electrolysis, or anything of that sort. There are certain occasions, however, where you may tap, and I will go into that presently, but the proper treatment for an ordinary

tumour is to do an abdominal section and remove it. I do not propose to go over the *technique* of ovariotomy. Go and see it done for yourselves. You will learn much more by seeing the thing done than by all I can tell you here.

I may mention that the percentage of mortality in ovariotomy is very low indeed, somewhere between 3 and 4 per cent. That, I think, is splendid; and why is it so? It is because we have learnt the proper antiseptic treatment of the abdominal cavity. We have learnt to be aseptic ourselves.

Now, the question is an interesting one as to when you should operate. The sooner the diagnosis is made, and the sooner the tumour is removed, the sooner the patient will be out of danger. If, therefore, I were asked, when should you operate? I should say, the sooner the better, when you are certain of it.

PREGNANCY.

Then you come to the question of pregnancy complicating an ovarian tumour. Is it not very curious that a woman with ovarian tumour or tumours, should become pregnant? Some women have become pregnant, having bilateral tumours of the ovary, what then is the effect of having an ovarian tumour upon the question of conception? Whether or not they interfere with the secretion and function of the ovary it is difficult to say.

Now we will take the question of treatment in a case of pregnancy with an ovarian tumour. A patient comes to you who is pregnant, and who has also a tumour which is a fairish size and the pregnancy is quite early—two or three months. What would you advise? I should advise you to operate on the tumour and not to interfere with the pregnancy. You have no right to induce abortion in such cases. Therefore, if the pregnancy is early and the woman has a tumour, I say operate upon the tumour, and leave the pregnancy alone. Why? For this reason. I do not think that the woman will necessarily abort. It would be very dangerous to interfere with the pregnancy, and not touch the tumour, because, supposing that, as a result of abortion, inflammation is set up and the tumour gets infected it would be dangerous. The danger is not so much with the pregnancy, therefore, but with the tumour. Now, supposing the pregnancy to be later—we will say six months—and with an ovarian tumour as well. What are you going to do then? That

is a little more difficult question. I think the right thing to do here is to wait until the child is viable. If the tumour suppurates in the meantime you cannot help it. You must wait until the child is viable and then operate on the tumour; for this cogent reason, the child being viable, if the woman does miscarry, it may live. And your operation need not necessarily interfere with the continuance of pregnancy even to full time, in fact, the probability is that she will go to full time. If we left the tumour and induced labour the chances are that it would inflame and become a source of great danger.

Now, we will take a case at full time. The question then arises, where is the ovarian tumour? Many of these tumours are above the brim of the pelvis, and then there is no obstruction to labour; but if you get a tumour low down in the pelvis, and jammed or wedged below the brim, there may be an insuperable difficulty to the passage of the child's head to deal with.

The next point to consider is, supposing you have a pelvic tumour of this sort, whether it can be pushed up. If you can push it up above the brim then it is all right, but if not then comes the difficulty. Supposing you have an ovarian cyst which you cannot push up, the question then is, would you be justified in tapping it *per vaginam*. I think I may say yes, for the time being, to get over the difficulty; but you must remember that you are tapping "in the dark," and you do not know what the cyst contains, and you may, therefore, get into terrible trouble. But in that particular case I think you might, within reasonable limits, tap the tumour. There may be another indication for tapping. In some cases in which you get enormous cysts, causing severe pressure symptoms in old people, complicated with bronchitis, bad dilated heart, œdema of the extremities, etc. Possibly, where it is not worth while, at the time, to subject such a case to the risks of a big operation. However, I have seen ovariotomy done in very old people who have got perfectly well. Tapping, however, is not a curative treatment, but merely a palliative one, and I do not advise it excepting in the cases I state. There is practically but one treatment for ovarian tumour, and that is removal. Sometimes you cannot do this, then incision and drainage must be done, but such cases are not nearly so hopeful, as you leave the primary source of the trouble still there.

THERAPEUTICAL NOTES AND FORMULÆ.

Ozæna.—Thorough spraying of membrane with some solvent of albuminous material; wash away this material with alkaline antiseptic solution; thoroughly dry membrane; apply remedial agent which will adhere to membrane, stimulating and irritating, establishing acute inflammation. When copious watery discharge has lasted ten days, use stimulating antiseptic solution. Peroxide of hydrogen solution (15-volume) best solvent for albuminous material; oil of mustard (gtt. vj–viij) in benzoinol or liquid albolin (ʒj) used with atomizer, forms admirable irritant.—*Med. News.*

Guinea-worm (Filaria Medinensis).—One compound sulphur lozenge (Garrod's formula) to be taken every four hours daily for at least ten days, then one lozenge three times a day during the next ten days, at end of which period cure; life-history and development of parasite having been cut short.—(*Indian Med. Rec.*)

Malakin.—Salicylic derivative, containing about fifty per cent. salicylaldehyde. Action is mild; no unpleasant effects, except for profuse perspiration. Of value in acute rheumatism, causing rapid amelioration of articular symptoms.
(*Münch. Med. Woch.*)

Acute Inflammatory Rheumatism.—

℞ Acid. salicyl. ʒ2⅛
  Potass. bicarb. ʒ2⅛
M. Place in mortar, dissolve in water until
    effervescence ceases, add—
  Liq. ammon. acet. ...   ...   f ʒ3¼
  Fl. ext. cascara sagrada arom. f ʒj
  Glycerini   ...   .   f ʒviss
  Aq. q. s.   ...   ...   ad f ʒxvj
M. Sig.: f ʒiiss every two to six hours;
    when patient perspires freely, every
    six hours.
(*St. Louis Courier of Medicine.*)

To Prevent Septic Infection through Abrasions.—

℞ Ichthyol ...   .   ʒj
  Flex. coll. ...   .   ʒj
M. Sig.: Apply with camel-hair brush once
    or twice daily.
(*St. Louis Courier of Medicine.*)

REVIEW.

A Treatise on Diseases of the Ear. By T. MARK HOVELL, F.R.C.S. Edin. (J. & A. Churchill.)   *Published at 18s.*

In a review intended for a journal of clinical medicine, and read chiefly by those who have to practise every branch of our constantly expanding profession it is a little difficult to choose the terms which we should apply to the work before us. In its completeness and in the clearness and accuracy of its information we know of no work to approach it; but its very exhaustive description of (we think) every form of trouble that has been known to affect the external, mid, or internal ear renders it at once a standard work of reference in time of serious doubt and difficulty rather than a familiar companion in every-day life. While speaking thus, we trust we shall not be mistaken in our meaning : the familiar symptom, the trivial operation, and the well-recognised condition are all there, as well as the rarities in complaints and appearances, and the fullest details of even the most delicate of aural operations, so that from the skilled specialist to the busy dispensary doctor the wants of all will be met. As specimens of what we mean, we may refer to pp. 261–268 as an illustration of the simpler operations, pages which have, by Mr. Hovell's kindness, already appeared in the "Clinical Journal"; while few but the most skilled experts would care to follow the details on p. 353 of the operation for removal of the ossicles.

Mr. Hovell must be very warmly congratulated on the production of such a complete work, so admirably illustrated, and so well printed. The wood-cuts are clear and excellent, sufficiently numerous without converting the book into an instrument makers' catalogue, and we think Messrs. Mayer and Meltzer well deserve this advertisement for the neatness and ingenuity displayed by them in carrying out the designs of many aural surgeons.

As a work of reference we can cordially recommend the book, and consider it will be found indispensable to every library of any pretension.

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

NON-TUBERCULAR CIRRHOSIS OF THE LUNG.

Delivered at the London Hospital, July, 1894,

By PERCY KIDD,

M.A., M.D. Oxon., F.R.C.P. Lond.,

Assistant-Physician to the Hospital.

II.

GENTLEMEN, — Before commencing the con-sideration of the subject of "non-tubercular cirrhosis of the lung," I want to demonstrate to you the case of this patient under the care of my colleague, Dr. Mitchell Bruce, by whose kind permission I am able to show him.

This young man's case is briefly this: He was in perfectly good health until August of last year, in which month he was one day sitting quietly in an arm-chair when he suddenly had a profuse hæmoptysis, bringing up about a pint of fluid blood. On two successive mornings he brought up a similar quantity of blood, and from that time he has had more or less cough and expectoration. In December last he had another hæmoptysis, which was still more severe. He brought up, he says, three pints on three successive mornings. Since that time his cough and expectoration have been more troublesome, and he has become weaker and short of breath. A fortnight before admission, which was towards the end of May, he had to give up work altogether.

If we inquire into his present symptoms, we find he has little to complain of beyond a little cough, without any expectoration. His nutrition is extremely satisfactory, he is gaining weight, his colour is good, but his fingers are clubbed. In fact, but for the history, we should not expect to find much wrong with him. Physical examination of the chest, however, discloses obvious signs of disease. I will not go at any great length into the physical signs, as you will have an opportunity of examining him for yourselves, but, roughly speaking, they are these. There is some flatten-ing under the left clavicle, with greatly diminished expansion. The impulse of the heart is felt as far out as the anterior fold of the axilla. There is dulness on percussion above the left clavicle; but from the clavicle down to the fifth rib there is hyper-resonance, while on the right side the percussion is normal. Percussing horizontally across the sternum we find the normal resonance of the right lung changing to hyper-resonance as we pass to the left of the sternum, until we reach the anterior fold of the axilla, when the note becomes less resonant, and in the axilla it is dis-tinctly dull throughout. At the back we find marked dulness over the whole left side, while on the right there is good resonance. On ausculta-tion, the breath sounds are normal over the whole right side, front and back. On the left side, anteriorly, the breath sounds are vesicular below the clavicle, but rather weaker than on the right side. Above the clavicle, and in the axilla, the breathing is extremely feeble, and when the patient coughs we hear a number of large, bubbling râles; and the vocal resonance in this situation is in-creased. Behind we have similar physical signs. The breathing is very weak over the whole left back; over the upper part, above the spine of the scapula, it is feebly tubular. On coughing, large splashing, or succussion râles are produced, very suggestive of excavation. On the right side the breath sounds are perfectly normal, and no ad-ventitious sounds are heard. The left shoulder, you will notice, is much lower than the right, and if we mark the position of the angle of the scapula on the left side we find it is lower than on the right, and closer to the middle line. He has, therefore, all the signs of a contractile disease of the left lung. Dr. Bruce, with whom I entirely agree, believes that the hyper-resonance on the left side in front is due, not to the left lung, but to extensive compensatory enlargement of the right lung bulging into the left side of the thorax. The physical signs indicate the existence of a much contracted left lung, containing a number of vomicæ, the heart being displaced to the left by the pulmonary contraction.

So much for the anatomical condition of the lung. What is the pathological diagnosis? Is

the condition tubercular or non-tubercular? Dr. Bruce thinks it is tubercular; I think so too; but I am aware that this diagnosis might very well be disputed, as positive proof is scarcely to be obtained in the absence of sputum. The reasons for regarding the case as tubercular are these. In the first place, consider the history. The patient was suddenly seized with hæmoptysis, having been previously in good health. That is not an uncommon history in chronic cases of pulmonary tuberculosis; but in my experience, it is not met with in non-tubercular cirrhosis. Again, the signs of excavation are rather more pronounced in the upper part than in the lower part of the lung. That is really the only important evidence available. The general condition of the patient, and the absence of constitutional symptoms, might seem to impugn the correctness of the diagnosis; but this favourable state of things is by no means incompatible with the existence of a very chronic tubercular process.

Turning now to the subject of

Non-Tubercular Cirrhosis of the Lung,

we find that a great many different names have been applied to this condition, with all of which I shall not trouble you, but I merely mention one or two old and well-established names: "chronic pneumonia," "chronic interstitial pneumonia," and "fibroid phthisis." I shall not again go into the question of the advisability of using the last term, having said sufficient on that point at the last lecture.

Let us now consider the different types the disease assumes. We may divide them, roughly speaking, into three:—

(1) The *massive form*, which is almost synonymous with the *lobar variety*;
(2) The *insular, broncho-pneumonic*, or *patchy* form;
(3) The *reticular* form.

(1) I will first say a few words about the massive form, and then show you sections illustrating some of the points I want to bring before you.

In this type, the whole or part of a lobe on one side is converted into a greyish, irregularly marbled, solid tissue of varying degrees of firmness. In some cases, which form a sort of group by themselves, the surface of the lung on section is finely granular. We shall see the importance of that point directly. Contraction of the lung is generally present to some extent, but it varies very

much in degree. Bronchial dilatation, or bronchiectasis, may be present, but it is not so common as in the next form, so we shall not say much about it, except that it may be either cylindrical or sacculated. But, apart from bronchial dilatation, a certain amount of destructive excavation is very common. Most of these cases of lobar fibroid induration of the lung, or lobar chronic pneumonia present foci of excavacation at some point. The cavities may contain pulmonary aneurysms, as in tubercular cirrhosis, though this occurrence is somewhat uncommon. Excavation may take on a gangrenous character in such cases. Emphysema is very common in other parts of the same lung, and on the opposite side. Fibrous adhesions of the pleura are almost invariably found. Acute pleurisy is uncommon, except as the result of septic changes in the lung, and in such cases should effusion occur, it is likely to be purulent.

The localization is an important point, especially with reference to diagnosis. In tubercular cirrhosis the process is limited to, or most marked at, the upper part of one or both lungs; whereas in the non-tubercular process, the consolidation is most marked over the lower lobe, and is nearly always confined to one lung.

Some writers have described a special variety of lobar cirrhosis; and it was to extreme examples of this class of case that Sir Dominic Corrigan gave the name of "cirrhosis of the lung." In these cases, one lung is greatly contracted in all its diameters, and converted into a mass of dense fibrous tissue, in which ramify a number of dilated bronchial tubes. That is what is called "Corrigan's cirrhosis," but I do not think anything is to be gained by making a special variety of it.

(2) A few words about the insular or bronchopneumonic type of pulmonary induration in which the lung contains patches of solid indurated tissue. The localization of these patches is not characteristic; we may find them in almost any part of the lung, but they predominate on the whole towards the base of the lower lobe. In this form of cirrhosis bronchial dilatation is rarely absent altogether, and bronchiectasis is apt to be sacculated. Emphysema is very common, and we may have practically the same accidents as in the lobar variety, the formation of cavities and pulmonary aneurysm.

The pathology of bronchiectasis is a point on which I may say a word—not that bronchiectasis

and cirrhosis of the lung are synonymous terms in any way, but bronchiectasis is so common in this affection that I think it well to say a word about it. There are various theories as to the development of bronchiectasis. Corrigan believed it to be due to the traction of the contracting lung drawing the walls of the bronchial tubes apart, the extension of the latter being favoured by atmospheric pressure. Another important factor, no doubt, is the positive pressure of cough, the effect of which is to produce enlargement of the bronchial tubes, especially if their nutrition is in any way impaired. It is probable that the condition of the bronchial walls has much to do with the production of bronchiectasis. The coats of the bronchial tubes are likely to be more or less softened, for being infiltrated with small cells, their elasticity is diminished and they yield more readily to the pressure of cough than healthy tubes. The inspiratory or negative pressure is concerned in the production of bronchiectasis to some extent. For if a large part of the lung becomes converted into a solid airless mass the result is that the other portions tend to undergo a compensatory enlargement, and in this way we get emphysema and probably bronchial dilatation also. These are the chief methods of production of bronchiectasis described. I do not believe that any one is exclusively true, but that all are operative in varying degrees in different cases.

(3) The last variety I have to speak of is the reticular, in which the lung is marked with a number of intersecting fibrous bands which give the appearance of a sort of trellis-work. Generally there is a good deal of pigmentation together with these fibrous bands, and the lung has a curious, slatey-grey, patchy appearance. The reticular cirrhosis is nearly always associated with emphysema and chronic bronchitis, and, during life, it is always unrecognised as far as I have seen ; in other words, it has really more pathological interest than clinical importance. The chronic inflammatory process beginning in the bronchial tubes is propagated to the peribronchial sheath, and the consequent induration follows the lines of the pre-existing connective tissue framework of the lung. I shall say nothing further about this variety, confining myself to the lobar and broncho-pneumonic types.

Of the histological changes I shall speak briefly. They consist in the main of an intra-alveolar and extra-alveolar growth of connective tissue. We shall begin with the intra-alveolar. In specimen No. 1, which I show you, we have a section of a lung which appears to be uniformly solid, but on a careful scrutiny, even with the naked eye, you will recognise the air sacs filled with a darkly stained material. Under the microscope, we find the alveoli filled more or less completely with a plug of fibrin containing a few colourless blood corpuscles, surrounded by a delicate capsule of a finely fibrillated tissue, containing a few spindle cells and round cells—in other words, a delicate sheath of connective tissue. Traces of the epithelium lining the air sacs can be seen here and there, but more often the investing cells have disappeared. The contents of neighbouring alveoli, in many instances, are connected by filamentous processes. At this stage the alveolar walls show little change. In the next stage the fibrin has disappeared, and in its place there is a little plug of young connective tissue, delicate fibres interlacing in various directions, spindle cells, round cells, and newly formed capillaries which are, in all probability, developed from the alveolar capillaries by a budding process. The pathology of such cases appears to be that the fibrin poured out during the acute stage of the pneumonia remains unabsorbed in the air vesicles, and acts like a foreign body, the result being that, in course of time, the fibrin becomes gradually organized and converted into connective tissue. Sooner or later the process extends to the alveolar walls and to the interlobular and peribronchial tissue. In other words, the cirrhosis diffuses itself along the normal connective tissue tracts of the lung. The bronchial tubes generally show signs of chronic bronchitis, desquamation of the epithelium, and cellular infiltration of the submucous tissue. There are also various other small points I need not allude to now.

In the extra-alveolar cirrhosis (of which I show you a good example, a specimen from a case of lobar pneumonia, which was followed by induration, the whole disease lasting about four months) the fibrous thickening affects the alveolar walls, the peribronchial sheaths, the interlobular tissue and the subpleural connective tissue, while any changes that take place in the alveolar cavities are of a secondary nature.

With this very brief exposition of the histological changes, I now pass on to what is of even more importance,—the etiology of these cases.

We may divide all cases of non-tubercular induration of the lung (excluding the reticular form)

into three groups :—(1) Those which are due to lobar pneumonia—that is, pneumonia in the widest sense, occupying a lobe, or more than one lobe; (2) those which are due to broncho-pneumonia; and (3) those arising from pleurisy.

A few words about the pneumonic origin of cirrhosis. Authorities are very much divided as to the existence of this form. No one denies that cirrhosis of the lung may be lobar, but some excellent authorities deny that this is the outcome of a true croupous pneumonia; and there is no doubt whatever that the sequence of events is extremely rare. The opponents of this view cite the fact that croupous pneumonia has remained unresolved for months, but that the patient has ultimately recovered completely, as proving that pneumonia has no irritative effect on the connective tissue of the lung. In answer to the objection that a good many cases are on record in which a fairly defined acute pneumonia was succeeded by a contraction and fibroid induration of the lung, as attested by post-mortem examination, they assert that these cases are quite exceptional, and are not to be regarded as instances of true croupous pneumonia. These arguments seemed to me very cogent, and I expressed a similar opinion about four years ago. Since then, however, further reflection and experience have forced me to the conclusion that, in exceptional instances, true croupous pneumonia must be regarded as giving rise to an induration of the lung. At the same time, there is much to be said in favour of the view advocated most prominently by Wagner and Heitler, that the pneumonia which gives rise to induration is not the typical croupous pneumonia. For, as they maintain, these cases do not begin, as a rule, with the same abruptness, the patient is not so severely ill from the first, and the course of the disease is subacute, or even "typhoid," according to Wagner. But then it cannot be denied that we see cases of what must be called croupous pneumonia, which terminate with a typical crisis in which there has been no well-defined onset. The patient has, perhaps, had a slight rigor, pain in the side, or simply a feeling of malaise, but has gone about his business for some days before he has consulted us, when, to our surprise, pneumonia is discovered. It has been pointed out by Heitler, that in cases of induration secondary to lobar pneumonia, retraction of the chest wall has seldom been pronounced, the reason being that these cases tend to end rather rapidly (as a

rule, within a few months), before there is time for contraction of the chest wall to take place. But it is probable that in other instances these pneumonic cases run a much more chronic course, become ultimately associated with contraction, and then present the features of "Corrigan's cirrhosis."

A word now about the existence of a special form of lesion in these cases of chronic lobar pneumonia, as it has been called. In a certain number of cases carefully recorded, the intra-alveolar changes, that I detailed to you, have been discovered; and this process has been well compared by Orth to the organisation of a thrombus. I have seen three or four cases myself in which this anatomical condition was present, and a fair number have now been recorded in medical literature. Dr. Auld, of Glasgow, maintains that in every case of indurative lobar-pneumonia the anatomical process is intra-alveolar in the first instance. This view appears to me to be too exclusive. For in some cases no intra-alveolar organisation can be recognised, and the change is essentially interstitial, as in one specimen shown.

If we could collect a large number of cases of non-tubercular induration of the lung—they are not common—we should find that the greater proportion of them belong to the broncho-pneumonic group. This class contains cases secondary to the broncho-pneumonia of measles, whooping cough, scarlet fever, and, probably, influenza. We have not even yet had time to see all the remote results of influenza, but I expect to find cases where influenza has been the starting-point of chronic pneumonia. In some instances long continued bronchitis seems to be sufficient to start this broncho-pneumonic form of induration. This, remember, is the form which, in the first instance, at least, is liable to be insular and patchy; but separate patches may, in time, become confluent and involve a whole lobe, or even large tracts of both lungs. Extreme cases may perhaps attain to the dimensions of "Corrigan's cirrhosis." As further causes we must reckon dust of various kinds, and any chronic lesions of the bronchial tubes. Thus, the pressure of an aneurysm, or of a carcinoma of the œsophagus may cause a localized fibrous induration of the lung, the outcome of a chronic broncho-pneumonia. Stenosis and dilation of the bronchial tubes are both efficient causes of chronic broncho-pneumonia and induration. A section I now show comes from the lower lobe of the lung of a child who died of broncho-pneumonia. The lung is riddled with a

number of small holes, which are all dilated bronchial tubes. Under the microscope you will find that the epithelium has entirely gone, while the bronchial wall is infiltrated with small cells, looking like a granulating surface. The infiltration extends into the peribronchial sheath, and is associated with patches of broncho-pneumonia.

Collapse of the lung is spoken of as a cause of induration of the lung, but the probability is that it is not the collapse, but the bronchitis which is so frequently associated with the collapse, that is to blame. In collapse from a simple pleuritic effusion it is quite exceptional to get induration of the lung.

Syphilitic gummata are probably to be regarded as anatomically broncho-pneumonic lesions, but they are very circumscribed, as far as my experience goes, and therefore clinically not to be recognised.

The last etiological factor to be mentioned is pleurisy. It is a factor which has been very much discussed. The late Sir Andrew Clark considered that pleurisy was one of the common factors of non-tubercular induration of the lung. Other excellent authorities have held the same view; but, as usual in medicine, there are as good authorities on the other side who entirely deny the sequence of events. I must say that my experience leads me to agree with those who deny that pleurisy is a cause of any extensive induration of the lung. In one of the sections shown you will see a so-called pleurogenic cirrhosis. The pleura is greatly thickened, and the adjacent interlobular septa stretching into the lung are also thickened. It is fair to assume that the pleurisy has been concerned in the production of these localized fibrous changes in the lungs, which in this case, as in others of the same nature, are limited more or less to the periphery, but I have never yet seen a case of extensive cirrhosis of the lung where there was any reason to believe that it was connected with pleurisy.

I have spoken at considerable length on this matter, but it is essential to have clear ideas concerning the pathology before we can have any rational conception of the clinical history of these cases.

We have now to consider the clinical history.

The onset of the disease may be acute, subacute, or chronic. Cases in which an acute onset occurs would mostly come under the pneumonic group. Usually the onset is subacute or even chronic. In a large proportion of cases the history is simply that of chronic bronchitis without any history of pneumonia at any time. The patients sometimes tell us that they have a chronic cough, and no further information can be obtained from them. But the chronic bronchitis has probably been from time to time associated with development of patches of broncho-pneumonia which have been imperfectly absorbed, and have laid the seeds of an indurative process.

The symptoms of the non-tubercular are very much the same as those of the tubercular cirrhosis, but one or two points I must mention. The expectoration is very liable in these cases to become offensive and putrid. This is an important point in the diagnosis, because in the typical, uncomplicated tubercular case the sputum hardly ever becomes putrid. In addition you may find in these cases that the expectoration has a markedly paroxysmal character. The patient brings up from time to time large quantities (perhaps half-a-cupful in five minutes) of a mucopurulent or dirty greyish foul fluid, and then, perhaps, for four or five hours he may be perfectly free. Where such paroxysms occur you nearly always find that the signs predominate at the base. The paroxysms, too, are largely related to alterations in the position of the patient. A very common history is that the patients bring up different sorts of sputum, sometimes frothy, at other times thick and offensive matter, the latter coming up in large gushes. On inquiry the patients may tell you that stooping down to fasten their boots or any sudden alteration in the position of the body seems to promote these gushes of secretion. These symptoms are pathognomonic of cavities or dilated bronchial tubes in the base of the lung. For contrary to what is seen in tubercular cases, where the fluids gravitate from cavities in the upper lobes towards the bronchi, and are continually being expelled, the secretions of a basic cavity keep accumulating till it is quite full. The cavity being lined with an insensitive pyogenic membrane allows the sputum to collect until it overflows into the relatively healthy bronchial tubes, and there excites reflex cough. Paroxysmal expectoration depending on postural changes can accordingly be easily explained by the tilting up of the basic cavity, causing the secretion to flow into the sensitive bronchi in its vicinity.

These patients have generally marked clubbing of the fingers, and become more or less anæmic.

Wasting, night-sweats, and fever may occur as in active tubercular disease, especially where bronchial dilatation or ulcerative excavation of the lung has developed. This complication carries with it a marked liability to certain septic accidents, especially to broncho-pneumonia in the healthy parts of the same lung, or its fellow, due to inhalation of the putrid secretion into distant bronchial tubes. Diarrhœa is uncommon, and may be caused by accidental swallowing of the sputum, or it may represent a septic symptom.

Professor Gerhardt, of Berlin, some years ago drew attention to the occurrence of severe rheumatoid pains in such cases, and attributed them to the pyæmic condition of the patient.

There is another complication to which these patients are specially prone, and that a very grave one—namely, abscess of the brain. It is probable that the abscess is embolic, depending upon absorption of putrid products in the lung.

One patient of mine died from a phlegmonous cellulitis of the skin of the abdominal wall.

Persistent suppuration is very liable to lead to amyloid disease of the viscera.

Another method of termination in these cases is by gradual enlargement of the right ventricle, death ensuing from slow asphyxia; but, so far as I have seen, the most frequent cause of death in these cases is septic poisoning of some kind in connection with cavities in the lung, or dilated bronchial tubes. The most common manifestation is a septic broncho-pneumonia. Pleurisy and pericarditis are very fatal complications, the former being the commoner of the two.

Physical Diagnosis.

If you remember what I have said about the anatomical and pathological aspect of the case the physical signs will readily occur to you. The diagnosis turns on the presence of contracting disease of the lower part of one or both lungs—mostly of one lung—together with the signs, in typical cases, of bronchial dilatation or cavities in the affected lung. We find flattening of the lower part of one side with displacement of organs. The heart is drawn over to the affected side, no upward displacement being noted, as in the tubercular variety. This, of course, depends upon the position of the contracting disease in the lower part of the lung.

I must warn you about some of the auscultatory signs in this disease, because unless you make yourselves familiar with some of their peculiarities you may overlook the presence of a large cavity or dilated bronchial tubes. You must not expect in these cases, even when there is marked fœtor and paroxysmal expectoration, to find invariably the classical signs of a cavity. In some cases you will be able to do so. But when, as often happens, the accumulating secretions fill the cavities and bronchial tubes you get signs almost suggestive of pleural effusion. There is dulness on percussion, absence or weakness of breathing, and no pectoriloquy, bronchophony, or adventitious sounds. But the picture may change in a few minutes. After a fit of coughing, ending with copious expectoration, you may suddenly detect cavernous breathing. These auscultatory variations are characteristic of cavities in the lower part of the lung. In addition to the cavernous signs there is often a peculiar adventitious sound of the nature of a rhonchus—that is to say, a musical sound, but unlike the typical sonorous or sibilant rhonchus, it possesses a peculiar croaking character. This, perhaps, depends upon the viscid nature of the secretion in the bronchi. At other times you may hear over these dilated tubes or cavities large, gurgling râles, or splashing sounds when the patient coughs, suggestive of air and fluid being agitated together.

I am only too well aware that this account of the physical examination is very imperfect, but I must now leave the subject and pass on to the

Diagnosis.

How are we to diagnose this condition, and which are the diseases liable to be confounded with it?

First of all, we have to distinguish it from tubercular cirrhosis of the lung. The two chief diagnostic points are the localization of the disease, and the examination of the sputum. Tubercle bacilli are to be found in the tubercular form and not in the other. In many cases all other criteria fail us, and we have to rely on this alone. When we have the greater part of one lung affected, and we have signs of contraction, and perhaps cavities, but the patient's nutrition seems fairly good, we may be in doubt whether the affection is tubercular or not, and the only safe way to settle the matter is by repeated examination of the sputum. In cases which follow an acute attack, the chronic pneumonia may be mistaken for the stage of resolution. But in the latter the patient's condition changes rapidly, although his lung may present marked

physical signs. The temperature falls, and there is a disproportion between the constitutional and the local signs, whereas in chronic pneumonia the pneumonic condition persists, the patient is still dyspnœic, and still has expectoration, cough, and pyrexia; his condition does not improve, and the physical signs change but little. Again, at first, there may be some doubt as to whether the patient is suffering from retarded resolution or from chronic pneumonia, and here the only point to rely upon is the progress of the case. In retarded resolution, although dulness, râles, and tubular breathing may persist, the general condition of the patient gradually improves.

Pleurisy, with effusion and empyema, may, in some cases, be confounded with this disease; but in the pleural cases with few exceptions, the side is not contracted, the heart is displaced to the opposite side, and the dulness is more intense, and the vocal fremitus is diminished. Errors of diagnosis are more likely to occur when there is no decided displacement of the heart.

Pleuritic thickening and adhesion may be mistaken for this disease; but although the chest wall may be much retracted the patient's general condition is good; fever is absent, there are no signs of bronchial dilatation or excavation, and vocal fremitus is always diminished.

Malignant growths have to be distinguished from this form of chronic induration of the lung. This will not often give you trouble, because new growths commonly lead to pressure on mediastinal structures. Among the most constant pressure symptoms I may mention enlargement of veins on one or both sides of the thorax. At times you may even have displacement of the heart to the opposite side, as in pleural effusion, though, as a rule, the amount of displacement is not great. The physical signs of new growth, too, are very much like those of pleural effusion. Enlargement of one side of the chest is not uncommon. Contraction is very rare.

Hydatids of the lung and liver may, in exceptional cases, give rise to mistakes, but it is hardly necessary to go into this diagnosis now.

Abscess of the liver perforating the lung is another possible cause of error. I have myself seen one or two cases of this sort where, without any obvious enlargement of the liver, the patient presented signs of disease of the base of the right lung, and expectorated purulent matter of a brickdust colour. But, as a rule, there are enlargement and tenderness of the liver, and signs pointing to the hepatic origin of the disease.

I can but briefly touch upon the question of prognosis. In the first place, we have to consider the characters of the sputum. Once fœtor has developed the patient is on the borderland of septicæmia, and his condition is grave. The persistence of bronchitis is an unfavourable symptom, and may prove ultimately to be a symptom of septicæmia, fœtor developing perhaps some time subsequently. As long as consolidation of the lung is not complicated with much bronchitis, or with fœtor of expectoration, and the constitutional condition remains good, the patient may live for years, otherwise there is a constant liability to accidents—broncho-pneumonia, pleurisy, pericarditis, cerebral abscess, hæmoptysis, and the development of amyloid disease.

Time will not allow me to go into the subject of treatment now, but there are one or two small points of which I should just like to remind you. The first indication should be to put the patient in some pure, sheltered atmosphere, such as that of the southern sea coast. In cases in which there is a certain amount of bronchitis, the treatment is like that of chronic bronchitis. But of more importance is the instruction of the patient, if he have bronchiectasis or any cavities in his lung, of the necessity of evacuating at regular intervals as much as he can of the putrid sputum. Get him to practise various methods, such as stooping down, and other changes of posture, which are successful in promoting expectoration. The more the secretion stagnates, the greater the risk of progressive bronchial dilatation and septic complications.

The last point I have to allude to is the surgical treatment—that is, puncture or free incision. Some years ago at the Brompton Hospital we were very enthusiastic about surgical treatment; but I am sorry to say the results have proved very unsatisfactory. Several cases promised to do very well; the fœtor disappeared rapidly after the operation, and the patient gained flesh, the sinus began to close, when suddenly cerebral abscess, or some other serious complication developed. Having had to assist at the obsequies of several patients who succumbed in spite of this treatment, I am not enthusiastic about it. The reason why we have been unsuccessful is, I think, that in these cases we have to do, not with a single cavity or dilated bronchial tube, but with many; and if we succeed in tapping one large cavity, there is always

a chance of there being one nearly as large which we cannot reach. Surgeons are now very shy of tapping these chronic cases. But if the affection begin acutely, with symptoms pointing to localised gangrene, the case is quite different, and there are several instances recorded in the medical journals in which free incision gave the best results. On the whole, however, one cannot at present recommend surgical interference in these cases, as the chances of relief are very poor.

A CLINICAL LECTURE

ON

EPITHELIOMA OF THE TONGUE.

Delivered at University College Hospital, Oct. 22, 1894,

BY

CHRISTOPHER HEATH, F.R.C.S.,

Surgeon to University College Hospital, and Holme
Professor of Clinical Surgery in University College.

GENTLEMEN,—We have had in the wards lately several cases of carcinoma of the tongue ; and it happens also that I have had a run of tongue cases in private practice. I, therefore, have here an unusual number of recent specimens of cancer of the tongue, on which I wish to make a few observations.

In the first place, with regard to cancer of the tongue, let me warn you against being misled by the cases which we see in hospital. As a rule the cases which come before us here, are well-marked examples of epithelioma of the tongue which can hardly be misunderstood ; and yet we find from time to time that mistakes are made—that cases are sent here as epithelioma which are simply gumma, while the last case I operated upon was one which had been treated as gumma, but it had undoubtedly been epithelioma for a good many months. The site of the epithelioma as a rule is at the side of the tongue, but of course there are exceptions. So also with regard to gumma, the site of gumma is usually in the centre of the tongue, but again there are exceptions. I have brought with me a drawing which I had made a good many years ago, in 1870, of the case of a patient who was then under my care with a well-marked ulcerated gumma on the side of the tongue. There can be no question about that

case, because he also had a syphilitic eruption on the thigh (a drawing of which I show), and because the case cleared completely up under the influence of iodide of potassium. There are no cases which are more satisfactory to the surgeon, to say nothing of the patient, than cases of extensive ulceration of the tongue, which clear up and get well under iodide of potassium. Of course as soon as they begin to heal you know that the patient is out of danger. But then let me say that there is also a little fallacy about these extensive gummata of the tongue, namely, that iodide of potassium will clear up a certain amount of the deposit, the deposit due to the syphilitic poison or to the chronic inflammation, but that there may be grafted upon that disease and left behind a small nodule of epithelioma, which may develop later on. I warn you of that, because unless you are aware of it you will be too prompt sometimes in saying that a tongue is going to get well under iodide of potassium, whereas, it is only the superficial inflammatory deposit which is cleared up, leaving the real disease at the bottom.

True cases of epithelioma occur generally after middle age. I have been looking up my private notes, and I find that the youngest patient on whom I have operated recently for cancer of the tongue was a woman, only 35 years old. Both circumstances are unusual ; in the first place that it should be a woman, and in the second place that she should be so young. All the other cases, the hospital cases and the rest of the private cases which I have had lately were in males, and they were all patients over middle age, some of them getting on into old age. Now, why does the tongue more frequently in the male than in the female develop epithelioma at the side? The explanation of this is partly due to the fact that men smoke a great deal more than women : their tongues are irritated by the tobacco and particularly by foul pipes. Again, the side of the tongue in both sexes is liable to irritation from the teeth. There is no question about it in my mind that one great source of carcinoma of the tongue is the constant irritation kept up by the fangs of decayed teeth. It is astonishing how people go on suffering pain, as they must constantly do, from the tongue being irritated by a sharp tooth, without applying for advice. There is just one other thing to bear in mind, namely, that even people who are careful about their teeth sometimes suffer from cancer directly due to the

apparatus they have had fitted into their mouths. A good dentist takes care that there shall be nothing to irritate the side of the tongue, but nowadays there are cheap advertising and very incompetent dentists, who not merely fit an apparatus into the mouth which is dangerous in itself, but an apparatus which cannot be taken out of the mouth. I need hardly say that everyone who wears artificial teeth should take out his teeth every night and certainly not sleep in them; partly in order that they may be cleansed, and partly that he may not run the possible risk of swallowing them in the night, an accident which happens from time to time. But those unfortunate people who have been to a cheap dentist, and who are wearing a very badly constructed plate which cannot be removed, get a collection of tartar, and irritation is set up and eventually (I have seen more than one case) develop carcinoma of the tongue.

Those well developed cases that we see in hospital are promptly recognized by the appearance of the ulcer, its everted edges, its characteristic induration at the base, and the fact that too often there is secondary enlargement of the glands. I need hardly say how glad we are when we find that there are no glands affected. No surgeon is justified in keeping a case of epithelioma under observation for a moment after he has made up his mind as to what it is, if the patient will consent to its removal. But every now and then we find cases of carcinoma of the tongue which have been diligently watched, not to the benefit but rather to the detriment of the patient, who by this time has enlarged glands in addition. The cases which puzzle us are of this kind. A man has that form of leucoplakia which you saw just now in a patient's tongue. He has chronic inflammation of the tongue going on for many years, the result very often of syphilis, and possibly of tobacco, and sometimes, if it is more than leucoplakia, it is called ichthyosis, of which I show you in this bottle an excellent example in a tongue that I removed from a patient at the end of September. There are two tongues in this bottle, and I happened to remove them both on the same day; the upper one from a private patient, and the lower one from a hospital patient. In the lower one you will see that there is a great exaggeration of epithelium on the opposite side to that which is ulcerated; in fact, it is leucoplakia developed into something very like ichthyosis. In the upper tongue you

will see that a good part has been lost by ulceration, and that the rest of it is by no means healthy, the cuticle being hypertrophied. The gentleman from whom I removed this tongue was under my care in the year 1885. He then had a tongue which I pronounced to be syphilitic, for which he then went under treatment, got apparently well, and I lost sight of him. His tongue became sore again, however, but he did not come to me until it had got into the condition that you see here. Both these cases, I am happy to say, have done remarkably well.

I show you a third tongue, which was also removed from a private patient, on whom I operated three months ago. That case is a curious one, and the history was this. Some few years ago this gentleman came under the notice of a physician of an insurance office. The patient is now 58 years of age, and the insurance was made some five or six years ago, when he showed his tongue, which was then in a condition of leucoplakia, not advanced at all, but still there were white patches on the surface of it. In consideration of that condition of the tongue the office did not refuse the patient's life, but raised the premium by a few years, and then heard no more of the matter. The patient, however, developed epithelioma of the tongue, and was practically dying at home. He was in a morbid state of mind and refused to see any surgeon, but at last his son persuaded him to see this same physician. He sent the patient to me, and I at once said, " There is only one thing to be done. You are being poisoned by the discharge in your mouth." I saw that he had enlarged glands in the neck, and, notwithstanding, I strongly advised him to have his tongue removed as quickly as possible. He agreed to that, and I removed the tongue three months ago. I saw him last week, and he was in a fairly satisfactory condition of health, but unfortunately the glands in his neck, which were already enlarged, were beginning to ulcerate and discharge.

These, then, are three examples of leucoplakia of the tongue which have developed into epithelioma in the course of time. I remember saying in a previous lecture of mine on Diseases of the Tongue that a case of leucoplakia was more liable than any other tongue to have cancer developed in it, although it was not by any means a certainty, and I say the same thing now. I would not remove a tongue simply because it had leucoplakia, but I should warn the patient that he must be very

careful with that tongue, and the moment any growth showed itself he ought to have it removed. If a tongue is in a more advanced stage of disease, such as the lower one in the bottle here (ichthyosis of the tongue), then I think there should be no hesitation at all. The ichthyotic tongue should be removed just as though it were ulcerated epithelioma.

I will now refer to the tongue which you saw me remove last Wednesday. You will remember that a man was admitted here, 49 years of age, apparently in good health, and having this story. He told me that his tongue became sore at the beginning of the year, and then, wishing for advice, he did as the public so often does—went to what is called a special hospital. Now it was said a good many years ago that the throat includes all diseases between the base of the skull and the levator ani, and, accordingly, this poor fellow with a sore tongue was treated at a throat hospital. He was also seen by a gentleman of eminence in London, who is an authority on diseases of the tongue, and both his own doctor and that gentleman agreed in giving him iodide of potassium, presumably regarding the disease he was suffering from as gumma. The patient did not get on, and somebody sent him to me. I saw him at my own house first, and went carefully into the case. I found a great mass of disease of the tongue, which I was quite sure was something very much more serious than gumma. Very likely he had originally a gumma in the tongue, but when he came to me a fortnight or so ago, there was evidently a large mass developed in the under part of the tongue, the outline of which it was difficult to make out thoroughly. It involved very seriously the tissues beneath the tongue, but there was no glandular enlargement that we could find. I explained to the class as we went round that it was not an ordinary case, but one in which it would be necessary to do a somewhat unusual operation. Those who were present last Wednesday will remember that I performed the operation which is generally connected with Mr. Syme's name —that I split the lower lip and divided the lower jaw with a saw, and then, holding the halves of the jaw well back, I was able to get round the growth, isolate the tongue and tissues beneath it, and remove the tongue, which you see here. Now I am happy to find that I went well behind the disease. You see the outline of the white deposit, which is epithelioma in all the muscles beneath

the tongue. The difficulty was on the left side, because there the tongue was attached very firmly, and I was conscious when I was removing it that I was in very close proximity to the tonsil. I mention this because it turned out at the post-mortem examination that the tonsil was infiltrated already with epithelioma. That, I believe, is a very unusual thing; in fact, I do not remember at this moment any case in which I have found the same occurrence. The operation, necessarily, was a very severe one. The man was nearly fifty years of age, and although we did everything we could to keep him going, he unfortunately died in the course of the evening, directly from the shock of the operation. That is just one of those events which you must be prepared for if you undertake serious operations of this kind. We did all we could to obviate the danger. The patient had brandy and hot water injected into the rectum before the operation, which is exceedingly useful in cases of very serious operation, particularly in connection with the mouth. If you amputate at the hip-joint, of course there is no difficulty in pouring brandy into the throat, but when you are operating upon the mouth, it is exceedingly difficult to do so, and for years now I have followed a hint that I obtained from an American surgeon: in all these severe cases to inject hot brandy and water into the rectum about half-an-hour before the operation. I generally put two ounces of brandy to four ounces of water, and have that injected. It gives a sort of reserve power which the patient can draw upon during the operation. In this case, after the operation we also gave the patient brandy down the throat, and injected ether under the skin. I am sure that the house-surgeon did all that possibly could be done, but the poor fellow died. There was a post-mortem examination, and here is the larynx. You will notice the stump of the tongue close down to the hyoid bone, and you will see the ligatures that I put on still holding on each side, and how completely I have got away all the disease, except in one particular. In connection with the left tonsil, there is, apparently, some little epitheliomatous development, which no doubt was caused by direct contact with the tongue, and which I rather suspected at the time of the operation.

I am glad to have the opportunity of speaking of this operation of Syme's, because it is one not often done. I have performed it a few times, but the cases in which it is required are not very frequent.

I very much prefer when I can to remove the tongue by the intra-buccal operation, and that is the operation which you see me usually perform. With regard to Syme's operation, I may say that it gives you plenty of room, and you can get well down to the hyoid bone, as you see in this case. There is no difficulty in controlling the hæmorrhage, and it is a satisfactory operation where the disease involves, as in this case, the whole of the sublingual tissues. This operation may be compared for a moment with the operation which you see some of my colleagues do, but which I do not like myself, namely, what is called Kocher's operation. That operation is performed by making an incision from the angle of the jaw to the hyoid bone and up to the symphysis, working in from the side of the neck until the mouth is opened, then drawing the tongue out through the opening. It is said of that operation that it has the great advantage that you can secure the lingual artery far back early in the operation, and also get rid of the glands. On the other hand, it is an extremely severe operation, and I know that deaths follow every now and then. Certainly it is an operation which lays the patient up very much longer than that through the mouth, for when an operation is performed through the mouth we very often send the patient out in a fortnight, certainly in three weeks. If there are glands to be removed, it seems to me that they can generally be removed quite as satisfactorily from the outside by a small incision without involving such important structures as Kocher's operation does. However, you must only take that as my opinion, and for what it is worth. I do not at all wish to influence you against what other surgeons think to be an extremely valuable operation.

Now let us come to the treatment of ordinary cases of cancer of the tongue. Make up your mind at once that nothing less than an operation will do any good. As regards any caustic treatment, tying the lingual artery or dividing the lingual nerve, they are not the least curative, though of course, they may do a little good by relieving pain. If you are to give permanent relief, or relief for a good many months, the only thing to do is to remove the growth. The operation which I generally perform is the intra-buccal operation, and if I am going to remove only one half I split the tongue down the middle. If I am going to remove the whole tongue I do not always split it, but perhaps take the tongue in its entirety, as you will notice in this specimen. With regard to this intra-buccal operation the difficulty is, of course, the hæmorrhage. If you look into the history of these cases you will find that surgeons used to be so nervous about hæmorrhage from the tongue that they very seldom operated upon it. It is curious on looking back, as I do now, for a good many years of professional life, to remember how few cases of tongue operation I saw as a student, and yet I was seeing the practice of Sir William Fergusson, who was then in his prime, and who was perhaps doing more operative surgery than anybody else in London. I find this class of case more common nowadays, and they are also attacked more readily, because we have found out how to control the hæmorrhage. The original way of operating upon the tongue, as Sir William Fergusson used to do, was with a bistoury. He used to draw the tongue out of the mouth, cut away the diseased portions, then pick up the vessel and tie it then and there. That does very well for operations in the front of the mouth, but if you have a disease involving the whole length of the tongue you will very soon find yourself in a difficulty if you attempt to operate by that method.

A good many years ago the *écraseur* came into use, and you will remember that that instrument carried either a chain or a wire. I dare say many of you have never seen a chain *écraseur*, as it is seldom used nowadays, and I therefore show one which was in use twenty years ago. It has a jointed chain, which was passed round the tongue, and then slowly, a link at a time, the chain was tightened, and eventually no doubt the tongue was cut through. It was a very troublesome and tedious operation, as it was very difficult to keep the patient thoroughly under the influence of the anæsthetic the whole of the time, and there was the great disadvantage that very often, notwithstanding the *écraseur*, there was hæmorrhage afterwards. The chain was superseded by the wire. I have here an *écraseur*, which is generally called the cold wire *écraseur* to distinguish it from the hot wire—that is a platinum wire which was made hot by passing a current of electricity through it. Some years ago the surgeons here were in the habit of using the galvanic *écraseur* rather extensively. It had, however, this great drawback, that one never could be quite sure of the battery. Just at the critical moment when you wanted to use the wire, although it had been

working beautifully up to that time, something went wrong with the battery, and it would not work at all, or worked very feebly; and even if it did work and burnt through the tissues there was another great drawback, that as a result of the application of the hot wire a slough was formed on the surface of the wound. That slough necessarily had to come away, and when it came away there was very often secondary hæmorrhage.

I may say, having used all these instruments, that it was most unsatisfactory to feel that you were leaving a patient with, perhaps, both lingual arteries divided, and neither of them tied (they might be satisfactorily twisted, or they might not), and that when you went out of the house, if it was a private case, you never could be sure that you would not be sent for in an hour or two with a message that the patient was bleeding, and you would have to go back. In consequence of those frequent secondary hæmorrhages occurring after the removal of the tongue, I found out for myself (and I think the teaching is nowadays generally

no very great anxiety afterwards. I am quite willing to allow that given a good light and a good gag, which is a very important part of the operation, and a good assistant, which is perhaps a less important part, you can manage to tie the arteries very comfortably in that way by snipping through the tissues slowly with scissors. Still even with everything, good assistant included, you sometimes get trouble through hæmorrhage.

Some few years ago I devised the method which you generally see me use now, of using a pair of strong angular forceps to grip the tissues, and then to pass a ligature round behind them. If I am removing an entire tongue I adopt the plan of dissecting underneath the tongue, dividing the sub-lingual tissues and the genio-hyoglossi muscles, and drawing the tongue forward until I have thoroughly exposed it and can see exactly where the section should be made. Then with a straight blunt bistoury I draw a line across the tongue (as you can see in many of the tongues here) at the point where I think I shall be well behind the disease.

CLAMP FORCEPS.

accepted) that you can arrest hæmorrhage from the tongue in a very simple way by putting your finger down the throat and hooking up with the fore-finger the hyoid bone with the stump of the tongue. I mention it particularly, because it is so exceedingly useful for a house-surgeon, and those who have to deal with these cases in the absence of the surgeon, to know that they can without the least difficulty stop all hæmorrhage if they will hook up the hyoid bone with the forefinger. They can then deliberately pick up the vessel and get a ligature upon it.

Secondary hæmorrhage hardly ever occurs now-adays. Those who operate through the mouth divide the tissues with scissors after the method recommended originally by Mr. Whitehead, of Manchester. They snip through the tongue, bit by bit, until they come down to the lingual artery, and then either pick it up before they divide it, or divide it and then pick it up. In that way a ligature is put on each lingual artery, and there is

Let me remind you that the corium, the superficial part of the tongue, is exceedingly tough; you cannot tear through it, but must divide it with a knife. The moment you have divided it you come upon the muscular tissues of the tongue, which are very soft and which can be readily torn, and if you have a fairly stout nail you can tear through them as deeply as you like. Next I slip my forceps up and clamp them on to the remains of the tissue in the line of section, first on one side and then on the other. Then I get a hemp ligature behind the forceps and, after sawing the tissues a little, tie it very firmly, and, lastly, with either knife or scissors, cut the tongue away in front of the forceps, which are then removed.

I may say that I began to use this method in the year 1891, and I have looked up my private cases to see how often I have performed this operation. I find one in 1891, one in 1892, one in 1893, and three in 1894. These, of course, are only private cases; I have not got an account of

the hospital cases, but they are more numerous, I know, than that. I must, therefore, have used this method certainly in a dozen or fifteen cases, and so far with complete satisfaction.

In the days when hæmorrhage was not properly controlled, and when patients were every now and then suffocated by the blood getting down into their lungs, a tracheotomy tampon was invented by a German surgeon named Trendelenberg. There was a tracheotomy tube with a little india-rubber ball covering it, which could be blown up so as to block the wind-pipe and allow no blood to pass down it. The objection to it, however, was that if it was blown out sufficiently to really make useful pressure it made the patient cough; and then the work had all to be undone. I have used it myself more than once, but I gave it up and found that we could practically get on better without it.

The after-treatment of an operation on the tongue was, a few years ago, in a very unsatisfactory condition. A great many of the patients from whom tongues were removed died in the course of the next week from septic pneumonia, and so bad was this that Mr. Barker devised a plan of doing tracheotomy, and then making the patient breathe through the tracheal wound in order that he might inhale nothing but sweet air. That, however, was very troublesome. It made the patient very miserable, and the tracheotomy certainly added a little to the risks.

What has revolutionized the treatment in these cases is the introduction of iodoform, which, as you know, is a magnificent antiseptic. You will notice that in all my tongue operations I dip my finger into the powdered iodoform and very freely rub it into all the tissues which have been divided. By doing this you will find that for twenty-four hours, at least, the mouth remains perfectly sweet. At the end of twenty-four hours it is well to begin washing out, but it is a great relief to the patient that nothing has to be done to him for those first twenty-four hours. After that we wash the mouth out thoroughly with an antiseptic lotion, and such a thing as a patient dying of septic pneumonia is practically unknown in operations on the tongue nowadays; whereas, if you look back to our records of some fifteen years ago you will find that a very considerable percentage of our cases died from that cause. I may refer you to a very able article by Mr. Barker in the last edition of the System of Surgery, a three-volume work written about that period, in which he shows how very great the mortality was from this form of septic pneumonia.

Now as regards results. I am sorry to say that one must confess that the results are exceedingly unsatisfactory, I mean as regards permanency. Every now and then we get a patient who survives an operation on the tongue for many years. Some of you will remember, no doubt, a man who was here a few months ago, whom I generally called the Great Jones, upon whom I operated in 1875 for a very extensive cancer, not simply of the tongue, but also of the front of the lower jaw, all of which I took away. Only a few months ago the man was here showing himself, and he was perfectly well. There was no doubt in that case that it was epithelioma, because there had been one recurrence already from a previous operation performed by another surgeon, and a microscopic examination also showed very conclusively that there was epithelioma. That, however, is quite an exceptional case, and it is rare to find a patient living nearly twenty years after an operation of that kind. I am sorry to say that the bulk of these cases do not live more than two years, and some of them do not live so long. You will find in the majority of cases that the recurrence does not, as a rule, take place in the mouth if the operation has been done thoroughly and satisfactorily, but in the glands, either below the jaw or in the neck. There is an example in the hospital at the present moment, that old man whom we saw just now, who had a very limited epithelioma of the tongue. You can see what a small piece of his tongue was involved, and yet before he applied for relief he had an enormous deposit in the glands beneath his left sterno-mastoid. There was no deposit in the glands below the jaw, but passing over this for some reason that I cannot explain, the disease affected the glands beneath the sterno-mastoid. These are the most fatal cases. When there are glands below the jaw I should strongly advise you to remove them, not necessarily on the day on which you remove the tongue, but a week or ten days afterwards. It is not a very difficult operation if you know how to do it, and the way is this:—Put your finger into the patient's mouth (I need hardly say have him gagged first) push up the gland from beneath the jaw with your finger, then make a very limited incision right down to it, and with a sharp spoon you can turn

the gland out with very little trouble. You must remember, however, that it is not the submaxillary gland which you want to remove, but the submaxillary lymphatic glands which have the deposit in them. They shell out readily enough if you do the operation in the way I have described.

The case is very different when the glands beneath the sterno-mastoid are affected. I know that sometimes very enterprising operations are undertaken for this. I read with admiration sometimes in the journals how a young surgeon has divided the sterno-mastoid, dissected out the glands beneath the carotid vessels, and all the rest of it. It is very pretty, but, in my opinion, it is not good surgery, because, undoubtedly, you cannot get away all the glands, and it is better to leave the patient alone. A certain percentage of patients die after these large operations on the glands, and I think, taking it altogether, that when the glands beneath the sterno-mastoid are involved it is better for the patient to leave them alone. He is certain to die, and he will not die any more quickly if you do not interfere, because it is practically impossible to get the whole disease away. There, again, I am only giving you my own opinion, and I know that other surgeons do not quite agree with me.

As a fact, patients who have been operated upon for epithelioma of the tongue very seldom live more than two years. I will take the list that I have here. I operated upon the first patient—the lady whom I mentioned—in 1890. She did not live two years. The next one was a gentleman, who had recurrence in the glands of his neck below the jaw, which I removed. He again had recurrence under the sterno-mastoid and I ventured to remove that. He was operated upon in 1892, and he did not live more than eighteen months. My next case is still alive, I am happy to say. I operated on him in 1893. He came to see me the other day, and he now has many of the glands beneath the sterno-mastoid involved, and I have advised him to let them alone. The old gentleman whose tongue I showed you here, who was sent to me by the physician of the life insurance office, was only operated upon in July, 1894, and already there is enlargement of the glands, which are breaking down and discharging, but still he is fairly comfortable. Another patient upon whom I operated in August, I am happy to say is well and has no glands. The patient upon whom I operated in September I saw this morning. He is fairly well, but the glands in his neck, which were enlarged on both sides when I saw him first, are very much the same.

Now, when I say do not remove the glands, I do not mean that they are to be abandoned altogether, because I am quite sure that by rubbing in iodide of potassium in the shape of liniment or ointment, you certainly do keep them in check, and it gives the patient something to do, which, after all, is satisfactory. When the worst comes to the worst, and they break through the skin, they discharge, and little pieces slough away, and the patient eventually dies—worn out by the cancer and by the discharge from the neck.

You must remember that cancer, either of the breast or the mouth, has a tendency to kill by lowering the vitality of the patient. These patients gradually get weaker and weaker, till at last they die; but I have never known a patient with cancer of the tongue who did not thank me for having removed it. A patient with an ulcerated tongue is in perfect and constant misery. The pain is excessive, the discharge is abominable, and it is impossible to give the patient relief. You get rid of all that trouble, however, by early operation. The proper way is to put it to the patient, and to his friends, and say, " I strongly advise you to have the tongue removed, because I think we may promise to get rid of all disease in the mouth, and probably you will have no further trouble in that region ; but I cannot promise that you will not have trouble in the neck, and eventually it certainly will shorten your life." One puts it in that diplomatic sort of way: you never say that the disease will kill the patient, but you say it will shorten his life ; and both the patient and his friends, if they have any sense, will understand pretty well what you mean. When the friends press you to know exactly what you are going to promise, I always say, " I do not think I can promise more than two years." That, I think, is the average duration of life. A patient may be lucky and go on a little longer, but it is the exception rather than the rule.

Hæmoptysis.—Rectal injections of chloral, 24 to 38 minims, repeating dose if necessary. Effect apparent in half or three quarters of an hour. Apparently acts as a prophylactic. It must first be ascertained if the heart is healthy.

(Gaz. Méd. de Liège.)

CLINICAL NOTES.

WITH DR. JOHN PHILLIPS
IN THE GYNÆCOLOGICAL DEPARTMENT
OF KING'S COLLEGE HOSPITAL.

Catarrhal Inflammation of Skene's Glands.

This young girl, aged 18, comes here complaining of cutting pains during micturition and when sitting down. On inquiry, we find that the pain came on almost suddenly four days ago, and that it is certainly getting worse ; she knows no cause for the trouble. Micturition is not increased in frequency, the pain is only in doing the act.

On exposing the vulva you observe at once the orifice of the urethra is not very marked, and that the lips are tumid and pale ; slightly below the orifice you notice two small prominences with reddish apices, one on either side ; on touching these she complains of exquisite pain. On passing a catheter, slight inconvenience only is experienced, and the water drawn off is quite normal. On pressing the urethral canal from above downwards, the finger being on the anterior vaginal wall, the apex of each of these little lumps is seen to exude a small drop of thick fluid, which has much the appearance and consistence of pus; a fine probe can be passed into the orifices, and travels about $\frac{1}{2}$ in. to $\frac{3}{4}$ in. along a narrow channel parallel with the direction of the urethra and running slightly posterior to it ; these are Skene's glands, so named after Dr. Skene, who first described them in 1880. This patient is suffering from acute catarrh of the lining membrane of these glands. The usual cause is gonorrhœa, but here, both from the patient's history and the healthy appearance of the vulva, we must look for some other etiology; probably " chill " would be as nearly as accurate as we could wish. As regards the treatment, I think sitting in a basin of hot water night and morning, with slight saline purgation, will suffice to relieve her ; if not, and the discharge becomes chronic, some intra-follicular application must be made in the shape of iodine or glyceride of carbolic acid, either by the insertion of the blunt point of a subcutaneous syringe-needle and injection of the fluid, or by applying the solution on cotton-wool wrapped round a fine probe.

Had this woman been married she would have suffered from great pain at any sexual approach. This disease also is one of the causes of "painful sitting," but painful micturition is not necessarily always attendant upon this condition.

Fibro-myoma of Uterus complicating early Pregnancy.

This patient is 28 years of age, and says she has been married only four months ; she thinks that she is much larger than she should be, and remarks have been made by neighbours as to her unusual size. She tells us she has menstruated only once since her marriage, and that was at the proper time, being normal in every respect. Up to two months ago her abdomen was perfectly flat ; she has felt no nausea, nor any fœtal movements. Before examining this patient, we must bear in mind that recently-married women will often wilfully deceive us as to their pregnancy, in order to hide the fact of intercourse having taken place before the ceremony. On examining her breasts we see a distinct areola with exudation of a drop of serous matter on pressure. The abdomen is occupied by a large slightly mobile hard painless mass of the size of a seven months' pregnancy. Its surface is smooth with a few rounded bosses laterally and at the fundus; no fluctuation or thrill can be elicited. On auscultation we find over the tumour nothing but a loud souffle synchronous with the pulse at the wrist; no fœtal heart sounds can be made out. There is dulness over the whole tumour and resonance in the flanks. *Per vaginam*, the cervix is distinctly softened, and moves bimanually with abdominal mass, the uterus proper cannot be distinguished, and there is no *ballottement*.

The patient is evidently pregnant, and if her word is to be accepted, about three months gone ; what, therefore, is the nature of the mass filling up the abdomen? As it was not there two months ago, we must bear in mind the peculiarity of fibromyomata when complicating pregnancy; this physiological condition seems in some way to act as a violent stimulus to their growth, and I can quite imagine it possible that this tumour (if it is fibroid) has rapidly attained to its present proportions during the past two months. It probably existed during the non-pregnant state as a small nodule on the uterus, and gave rise neither to hæmorrhage nor pain, and therefore the patient's

attention was never attracted to her pelvic organs, and, but for the size of her abdomen, she would not even now have asked our advice.

Assuming that this growth is a fibromyoma complicating an early pregnancy, what are we to do for the woman—is she to be allowed to go on, or are we to interrupt her pregnancy? It is perfectly evident to you from the great distension already present, that allowing her to go to term, or even to seven months, would be entirely out of the question, provided growth is as rapid as it has been heretofore. I should, therefore, strongly advise her to come and see us in a month's time; we can then compare our measurements made then with our present ones, and if we find the tumour still rapidly increasing, induction of abortion should be produced without delay. Remember, however, that this is a dangerous proceeding, post-partum hæmorrhage, retention of the decidua, sepsis, and pathological changes in the fibroid tumour may each and all take place, jeopardizing the patient's life. In favourable cases, after abortion has been completed, the tumour begins to atrophy and may almost entirely disappear, to be aroused again into similar activity should pregnancy recur.

Climacteric Hæmorrhage.

This patient, who is now 55 years of age, you may remember seeing about a year ago, when she applied here for profuse and repeated hæmorrhages; you may recall the fact that I urged her to become an in-patient, so that an accurate diagnosis might be made, as her symptoms were suspicious of commencing malignant disease of the body of the uterus. The interior of the uterus was explored, and nothing found. She has been here occasionally since, and to-day you hear she says that she has the hæmorrhage at much greater intervals—as long as two months elapsing without any loss occurring; she is also making flesh, is robust and well-looking, and complains now of nothing beyond involuntary flushes and severe occipital headache. On examining the uterus you find that it is quite small and mobile.

This patient is suffering from the hæmorrhages which often occur at the menopause; it is hardly necessary to impress upon you the importance of finding out as soon as possible whether any morbid growth is present to produce this loss; excluding these, and an ill-understood condition called

hæmorrhagic senile endometritis, we can come to the conclusion at which we have arrived, with every feeling of confidence, and advise the woman to wait patiently, and the hæmorrhages will gradually cease.

Atresia of Os Uteri Externum.

This woman is 54 years of age, and has not menstruated for six years. She comes here complaining of backache and bearing-down pain; there is no discharge. On vaginal examination, the uterus is small, the cervix bulky, and indicates no depression, corresponding to the external os uteri; on exposing the cervix by means of a Sims' speculum, nothing but a smooth surface can be detected. Pressing firmly with the point of the sound, about the situation where the os uteri externum should be, you notice it has suddenly passed into a cavity about an inch in length; on withdrawing it, about half-a-teaspoonful of clear mucus escapes.

The cavity is that of the cervix, and the atresia is one of the local indications that the menopause is established, or in process of being produced; sometimes the internal os uteri may become closed in a similar manner, the cervical canal then becomes a slightly dilated closed sac, containing healthy mucus. No treatment in either case is necessary.

FORMULÆ.

Catarrhal Conjunctivitis.—Wash, to be used four or five times daily :—

Sterilized water	...	...	... Oij
Opium extract	...	...	... ℳ i$\frac{3}{4}$

For instillation morning and evening :—

Distilled water	...	...	... f ʒiiss
Nitrate of silver	...	...	... gr.i$\frac{3}{4}$
Rousseau's laudanum	...	...	gtt.v

Filter. When suppuration has diminished, replace by distilled water, f ʒiiss; sulphate of zinc, gr.$\frac{1}{6}$.—(*L'Union Médicale.*)

A CLINICAL LECTURE

ON SOME

ELEMENTARY POINTS IN THE DIAGNOSIS of NERVOUS DISEASES

ILLUSTRATED BY CASES OF

LEAD PALSY, LOCOMOTOR ATAXIA, DISSEMINATED SCLEROSIS, AND ACUTE MYELITIS.

Delivered at the Sheffield School of Medicine, Oct. 30, 1894,

By ARTHUR J. HALL,

B.A., M.B. Cantab., M.R.C.P.,

Hon. Physician to the Sheffield Public Hospital; Lecturer on Physiology at the School of Medicine.

GENTLEMEN,—The subject of my lecture to-day is a very wide one, for I am about to show you five cases of organic disease of the nervous system, which include at least three different affections, in no way related to one another, except in so far as they are all affections of the group of cells forming nervous tissue. It is not my intention to read you any full notes of these cases to-day, as you can at any time obtain them from the wards for yourselves, and I wish rather to point out from these five cases some general features of organic nervous diseases.

The first case is that of a young man, aged 23, who was admitted on September 2nd, 1894, complaining of loss of power in his right arm, which began four weeks before admission. During this period it had gradually been getting worse, and on admission, the following condition was found. He was unable to extend the right fore-arm on the arm, or the hand on the forearm, and was therefore quite incapable of doing his daily work. On inquiry, it was elicited that he was a file-cutter by trade, and that during the last few years he had suffered from the various cardinal symptoms of chronic lead poisoning, or plumbism, to which this trade exposes its workers, namely, headache, constipation, and colic. And I may here explain to you how this occurs: the file is laid upon a block or bed of lead, so that when one side of the file is cut, and the file turned over

in order that the opposite side may be cut, the side already cut may have a soft material to rest upon, and not be spoilt by the force used in cutting the opposite side. As you know, the cutting is done with a heavy hammer and chisel, and the constant hammering causes the block of lead to be worn down, so that the fine lead dust gets on the worker's hands and fingers, and so on his food; and it is this constant daily swallowing of small quantities of lead which brings about this most serious pathological condition of lead poisoning. Leaving aside the general symptoms of plumbism, let us consider how we are to diagnose the seat of the lesion in this case. If we examine more carefully the exact muscles which are affected, we find that they are those supplied by one nerve, the musculo-spiral nerve, and moreover there is distinct tenderness on pressure over this in the arm, and over its branch in the fore-arm, namely, the radial nerve: there is also some vague loss of sensation, or anæsthesia; but it is very slight and ill-defined. Now, the question before us is, Where is the lesion? It is evident that, theoretically, it might be in the muscular fibres themselves, or in the nerves going to them, or in the spinal cord, or higher up still in the brain itself. For lesions in any of these places may cause paralysis. How, then, are we to set about diagnosing its seat? In the first place, the fact that only muscles supplied by one nerve are affected, would be very curious if the muscles themselves were the seat of the lesion, for they do not all lie side by side, but are spread over the whole length of the back of the arm; moreover, they react well to electricity; there is also some slight loss of sensation, and tenderness over the nerves, which would be unaccountable if the muscular fibres alone were affected. So we may fairly dismiss the first position for our lesion, namely, the muscles themselves. Let us take, secondly, the spinal cord, leaving out the nerve-fibres themselves, for the present. Is the lesion a spinal lesion? Now the only place that such a lesion could be, in order to produce a paralysis affecting the muscles supplied by the musculo-spiral nerve only, is the ganglion cells of the anterior cornu of grey matter, from which the fibres of the mus-

culo-spiral nerve arise, for at any other point in the central nervous system the further connections and distributions of these have got so widely spread that no series of lesions could conceivably pick out these, and these only, to the exclusion of anything else.

And this statement will also exclude the possibility of the disease being situated higher up in the cord or in the brain, for the arrangement of fibres as they pass up to the motor area is no longer one of distinct anatomical spinal nerves, but of groups of fibres connected with muscles whose actions are associated together. Is the lesion then in the ganglion cells of the anterior cornu of the grey matter from which the fibres of the musculo-spiral nerve arise? There are two reasons, one anatomical, the other clinical, which seem to me to answer the question in the negative fairly clearly. You will all remember that the musculo-spiral nerve is formed by the posterior divisions of the 5th, 6th, 7th and 8th cervical nerves in the brachial plexus, and, therefore, the lesion would necessarily have to affect ganglion cells scattered over a length of grey matter extending from the origin of the uppermost fibres, opposite the 5th cervical vertebra, down to the origin of the lowest fibres from opposite the 8th cervical, that is, a distance of some two inches, and yet not touch any of the other numerous ganglion cells situated close around, and giving off the fibres to the rest of the brachial plexus. Such a lesion is, to say the least, improbable. The clinical objection to this being a lesion of the cells of the anterior cornu is still more weighty, namely: that in diseases of these cells, there is wasting of the muscles or atrophy, which is absent in this case. Moreover, it would not account for the tenderness over the nerve-trunk or the slight anæsthesia. Please remember that I am not discussing paralyses due to lead; in general, they differ very greatly in different cases, and possibly in the seat of the lesion. I am merely quoting this case.

We are left, therefore, with only one position for the lesion, namely: the peripheral nerve itself, and this is confirmed by the tenderness which we find along the course of the nerve This is a case of peripheral neuritis of the right musculo-spiral nerve, due to lead poisoning. If you ask me why the musculo-spiral nerve trunk is especially and solely affected, I cannot say. You will notice there is not complete anæsthesia over the cutaneous areas supplied by the

musculo-spiral, although there is complete paralysis, explicable on the supposition that sensory or afferent fibres are not so easily affected as motor or efferent, and recover more readily. Perhaps the efferent or motor fibre has a more elaborate arrangement in order to act under the voluntary will, and is more easily deranged, and less easily mended. After complete division of a mixed nerve, causing complete paralysis and anæsthesia, the reunion of the nerve is heralded by the recovery of sensation, which precedes the recovery of motion. I have been tempted to go into the method of thought which should be adopted by each of you when trying to diagnose a case of organic nervous disease, because I know how apt a student is to try to diagnose nervous diseases without the least thought, hopelessly bewildered by the innumerable long names attached to them. Do not rely on the presence or absence of the knee-jerk, to the exclusion of all your anatomical and physiological knowledge, in making your diagnosis of a nervous disease, it has its own proper value, and no more.

The next two patients I shall show you are suffering from the same disease, and curiously enough they both came up to my out-patient department during the same week. They are both men, aged 40 and 50 respectively; both were employed in the same works, and in both there is a probability that they have had syphilis, though the history is not clear. [Patients introduced.] Their chief and most obvious symptom is an inability to walk straight, there is no evident loss of power in their legs, but they cannot manage them properly, and this difficulty becomes more obvious when they have to perform any movements requiring rather more than usual nicety of adjustment. Thus, on turning round, or walking between narrow limits, the difficulty is much more accentuated. In other words, they have lost the power of co-ordinating their muscular actions. And this is not limited to their legs only, for you will find that they are also incapable of performing delicate movements with their hands, such as picking up a pin, buttoning their clothes, or counting money from one hand to another. If we were now to go through the same mental process of excluding different parts of the nervous system, we should find that though not quite as simple as before, their lesion is primarily a disease of the afferent tracts of the spinal cord—not the motor or efferent; for you will observe that if I ask them to perform any of

these movements with their eyes shut, so that no visual sensations help them, they are absolutely unable to guide or control their muscular contractions at all. They cannot even stand up by themselves, or do anything delicate with their hands. Moreover, they have lost sensation chiefly in their legs, although it is not quite abolished. This is evidently not a disease of the muscles themselves, or of the nerve trunks, for there is no paralysis, neither for the same reason is it a disease of the anterior cornua. It is, however, a very definite disease, not uncommon, and from pathological researches we know that it is due to a lesion of the posterior columns of the spinal cord, which we call Locomotor Ataxia or Tabes Dorsalis. The primary defect is an insufficiency of afferent impulses informing the centres of the exact amount of work which each muscle is doing, information which is absolutely necessary for the accurate adjustment of those muscles. You may call this the muscular sense or not, as you like, but it undoubtedly is something more than the mere sense of touch. Directly you take away from such patients the aid of vision, so that they cannot see what their muscles are doing, they become infinitely worse. The many other associated symptoms and signs in these cases I must pass over to-day, and show you another closely allied case.

This patient, a married woman, aged 42, had the misfortune to acquire syphilis fourteen years ago, and for the last four years has suffered from gradually increasing difficulty in walking and in using her hands, she is now hopelessly bedridden, and presents very curious symptoms. In many respects she resembles very closely the last two cases I have shown you; thus, she cannot control her muscles when using them, and becomes worse when visual aid is removed; and she has lost the sense of touch also. But besides these defects, she has completely lost all sense of pain, so that you can stick a pin into her anywhere without her feeling it, and this although she can tell hot from cold. Another interesting feature about her which was not present in the last two cases is that she has violent tremors on attempting to use her muscles, becoming more wild and unmanageable the more she attempts to continue the actions, and with it all a great loss of power. Looking at her eyes you will notice that she has paralysis of various ocular muscles also, pointing to a lesion or lesions at the base of the brain. Here, then, we have a very widespread affection which evidently involves

the whole extent of the central nervous system. In many features it closely resembles locomotor ataxia, but the peculiar violent tremors, which even occur in the ocular muscles producing nystagmus, or oscillations of the eyeball, point more to a widely-spread disease which we call disseminated sclerosis.

The last case I shall show you to-day is an acute disease of the spinal cord, occurring in a little girl, aged 12 years. Twelve days ago she was " marching " at school, when she fell down, felt queer in her legs, had to be carried home, and in four or five hours her legs were completely paralysed. She could not control her urine or fæces, but must pass them immediately she felt a desire to do so. She has been the same ever since. She has no pain, nor can she feel any pain on pricking her below the level of the anterior superior spines of the ilia. She can feel when touched below this level, but only imperfectly. She has got bedsores on her buttocks, which are painless. She has entirely lost her knee-jerks; there is no wasting of the muscles.

Pay particular attention to this little girl's bedsores; they are very important in making our diagnosis, for they are acute. The last three cases I have shown you have been lying in bed for months without getting sore at all, and this little girl is quite sore in less than a week. If you follow out the method of reasoning I have pointed out to you, you will see that she must have an affection of the spinal cord, somewhere about the level of the upper lumbar region, which, as power and sensation are both affected, must stretch transversely across the cord. It is, however, not completely blocking the whole cord, for she can feel a little. It is a case of acute incomplete transverse myelitis.

We have now seen five cases of organic nervous disease—one in the peripheral nerves only, three in the spinal cord, and one in the spinal cord and brain. They differ very widely, but we may still compare their causes, and the possible results of treatment to the patients.

Ætiology.—In no less than three of the cases, syphilis is the primary cause, certainly in two out of the three, and probably in the third also. In the fourth the absorption of lead into the system, and in the fifth, viz., the little girl, nothing has as yet transpired to account for it.

Notice also, that in the three cases due to syphilis, the disease is slow and chronic, being

counted by years, whilst in the other two cases it is acute, being counted by weeks in the one case, days in the other. The chronicity of the syphilitic nervous lesions is on the whole characteristic of tertiary or late syphilitic lesions. The absence of any evident cause in the case of acute myelitis is also usual; sometimes there is a history of getting wet through, or considerable exposure, but often none.

Prognosis.—Unfortunately, we may say at once that for the first three cases due to syphilis, there is no hope of improvement under treatment. These diseases do not yield to anti-syphilitic remedies as a rule, and in all these cases they have been tried long and frequently without benefit. Nor will any other drug or method do any better, you can only relieve symptoms as they arise with the ordinary means in your power; promote appetite, assuage pain, relieve constipation. But they may all three live many years yet, and eventually die of some inter-current disease, such as pneumonia.

As regards the case of plumbism, the prospect is much better, already the patient is regaining power in his arm, under a course of iodide of potassium, coupled with regular daily application of the electrical current to the paralyzed muscles. But he will be unable to continue at his own trade, for fear of further trouble from the lead. Fortunately, he is young, and can probably find another means of gaining a livelihood. As to the little girl with acute myelitis, the future is not hopeful. Whether she will recover the use of her limbs is extremely doubtful, and how long she will even live is very uncertain; she is threatened with troubles from her bedsores and incontinence, which prevents her being kept dry, and her position is very precarious. In her case the nurse is of far more importance than the doctor, until the future declares itself.

N.B.—Since the delivery of this lecture there has been considerable recovery of power in the flexors of both legs.

Antipyrin in Cystitis. — Inject $2\frac{1}{2}$ to 5 drachms of 4 per cent. solution, allowing liquid to remain ten minutes in order to be absorbed. If bladder be distended, wash out and inject from 2 to 4 ounces of 1 to 100 or 1 to 200 solution, leaving it in bladder. Causes relief of pain, acts as antiseptic, and may be used for months.

(VIGNERON, *Concours Médical.*)

CLINICAL NOTES.

WITH DR. JOHN CURNOW AT KING'S COLLEGE HOSPITAL, LONDON, SUMMER SESSION, 1894.

THIS woman, aged 54, is a typical example of old cardiac disease. She has had a very arduous life as a village nurse. She has had no rheumatic fever. For the last six months she has suffered from palpitation and shortness of breath. She was sent in with a little albumen in her urine, and was considered to be ill from renal disease. The first noticeable feature in her case is that the apex beat of the heart is displaced downwards and outwards; but there is no heaving impulse, or other sign of cardiac hypertrophy. The dulness is increased laterally as well as downwards and outwards; presumably, therefore, the left ventricle is enlarged, as also is, evidently, the right ventricle. There is, then, a condition of general dilatation without hypertrophy. No thrill is detectable. On auscultation there is heard at the apex a systolic murmur conducted to the left. At the base there is also a systolic murmur, much rougher than that at the apex. A faint diastolic murmur can also be made out there. The patient has a very small pulse. There is some œdema of the legs. There is a trace only of albumen in the urine. The liver dulness is not increased. The patient has markedly improved since admission, the treatment having consisted of iron, digitalis, and, lately, a little arsenic. She has, also, of course, had rest in bed and good food.

The question now is what exactly is the condition of her heart? and what are the chances of her recovery? There is no doubt whatever that in addition to the valvular mischief she has dilatation of the cavities, not to an extreme, but still to an appreciable degree. She has no compensatory hypertrophy, and all her symptoms are those of a weak heart—viz., dyspnœa, palpitation, œdema of the legs, and a slight amount of albumen in the urine. She has had no rheumatic fever, but she has been very hard-worked,; presumably, therefore, the valvular lesion is due to chronic atheroma, not only of her mitral valves, which are most affected, but also to a certain extent of her aortic valves, and probably of the aorta also.

What are the chances of this woman's longevity? No one can tell at what time this condition first

came on or for how long it has been silently progressing. She must have had some atheromatous change for a considerable number of years, without, perhaps, the incompetency of the valves. If she had no hard work to do, and could take every care of herself, so that she would not be exposed to adverse climatic influences, she might go on for a long term of years yet, as many people with chronic valvular lesions, consequent on atheroma, have done. The question with her is whether she can attend to her duties as a village nurse, whether these will be very onerous, and what will be the amount of exposure to which she will be subject? Taking into consideration the prognosis in a case of heart disease, what you have first to deal with is the valvular lesion, next, the condition of the walls of the heart—whether there is compensation going on or not—and then the conditions of the patient's life. Each of these points will seriously affect the prognosis. In this case there is no regurgitation of any consequence through the aortic valves, no painful distress, and the probability therefore is, in fact there is every certainty that she will not die suddenly from syncope or angina. In the next place, she has no hypertrophy to drive the blood with too much force into the arteries, so that she probably will not rupture a blood-vessel in her brain; but owing to the fact that the dilatation is without any attendant hypertrophy, the chances are that she will die simply of asthenia from her inability to get her living without effort and strain. There are only two factors really to be considered therefore—the hardness of her life and the weakness of her heart. The treatment must, in view of this, be entirely a sustentative one, with good food and rest. There was much improvement while in the hospital. She has given up village nursing, and her present condition is now (November) most satisfactory.

2. H. M., a man, aged 48, came in on May 12th, stating that four days previously he had been seized with an acute shivering fit accompanied by pain in the head, which was so severe as to prevent his sleeping. In addition, he had extreme sickness three or four times previous to coming to the hospital. He was intolerant of light and noise, and had had slight delirium. On admission he was suffering markedly from pain in the head, particularly at the back; he had pain all down his spine with retraction of the muscles at the back of the neck. He could not be lifted in bed, but had to be turned on his side when examined, on ac-

count of the rigidity of his spine. His tongue was coated with a very thick white fur, and his breath was very offensive. His temperature was 101° F. The pulse was slow, compressible, and only 66.

There was no doubt whatever, in the first place, that he was suffering from some acute infective disease, and the question was what this was likely to be. Although his pulse, being only 66, rather negatived the idea of typhoid, we looked carefully for typhoid spots, but found none. Of course, typhus is almost unknown in London, and we scarcely expected to find that, and this was after mention eliminated from the diagnosis. The next idea was that, although the disease is extremely rare, we might have here a case of cerebro-spinal meningitis. It reminded me vividly of some cases I had seen twenty years ago when I was house-physician during an epidemic of cerebro-spinal meningitis in London. I looked at his discs. The right was certainly blurred a little, as was also the left, though not so markedly. In addition his abdomen was retracted. These pointed to the possibility of a cerebro-spinal meningitis in an early stage. He was given a simple purge, and no other medicine was administered. The temperature came down in three days, and, with the exception of some stiffness in the back and some rigidity of the muscles of his neck, he is now quite well.

The question arises as to whether this could by any possibility have been a case of cerebro-spinal meningitis. In the first place, cerebro-spinal meningitis is generally epidemic. Secondly, there was no epidemic present at that time. In the third place, on the fringes of an epidemic of cerebro-spinal meningitis you meet with abortive cases like this, but the true epidemic cerebro-spinal meningitis is one of the most fatal diseases that can possibly occur; and it kills rapidly or leaves strong definite after-symptoms. If, therefore, this is a case of that disease, it is one of the abortive forms. My opinion, however, is that it is a severe attack of influenza from which this man has suffered, one of the worst that I have seen, and the only one I have seen running this very acute course. I have seen them with pain in the head, back, and so on, leading one to imagine that the brain and spinal cord were involved, but nothing like any definite, special local lesion. And this is one of the cases which I should put forward very strongly as bringing severe influenza and some of these other maladies of whose

etiology we know little or nothing, like epidemic cerebro-spinal meningitis, almost into one category.

We are now giving him a little quinine, and will begin allowing him some food. (He went out quite well after being a fortnight in the hospital.)

3. This man, aged 34, came in on March 21st, and went out on April 14th, but was readmitted on May 8th, after having been partly at a convalescent home, and partly at work in the interval. He is a railway porter, and much exposed to wet and cold. Three years ago, he was under treatment at the London Hospital, where he was told he had Bright's disease. You will notice first how extremely pale he is. In the next place he has had some dropsy of his feet and face, but that has disappeared under rest. He has a slow, incompressible pulse, with the signs of hypertrophy of the heart, his apex-beat being an inch below the nipple. The cardiac dulness is increased. The peculiar feature of the case, however, is that a loud abnormal sound can be heard over the base of the heart, particularly over the big vessels. It is not conducted; it is very loud, and is increased by pressure with the stethoscope. The special question we are discussing is its nature. The hypertrophy which is shown by the displacement of the apex-beat downwards and outwards is also shown by the heaving impulse which is quite perceptible to the eye. Of course that might mean that this basic murmur is aortic, or it might simply be a double sound caused by the stress of the circulation, as a result of the renal disease. I have no doubt however that it is an exo-cardial sound, *i.e.*, pericardial, first, because it is so limited in its area; secondly, because it is over the common site where we hear rough friction sounds; and thirdly, because it does not affect the pulse in any way, whilst an aortic lesion almost always does. But chronic pericarditis is comparatively rare in renal affection; for generally in renal cases acute pericarditis supervenes, and, of course, this leads to a very rapid end.

In addition to the condition of the patient's heart, I want also to call your attention to his urine, which is so absolutely typical of renal disease, that you are tempted to be able to make a diagnosis almost by the mere look. In the first place, it is very pale; in the next, it has a very low specific gravity; and in the third place, you have an abundant deposit which is evidently organic in character, by which I mean that it does not consist of ordinary urates, or phosphates. On testing there is found to be present a very large amount of albumen—two grammes to the litre. Under the microscope are seen granular casts: some containing oil, and others merely hyaline.

The ordinary treatment is to sweat the patient and purge him, to give him iron and keep him on a milk diet. That, of course, will prolong his life; but the prognosis is absolutely fatal when you have casts such as are present here. Whilst you have epithelial or granular casts, or even small hyaline casts, you may be hopeful that the case may improve; but immediately you begin to get large hyaline casts, or large fatty casts, like these, and such a typical appearance of the urine with a low specific gravity, and a large amount of albumen, there is no question as to what the result will be in no long time. Of course, with the onset of any concurrent acute illness the patient will die from that disease, as *e.g.*, were he to have typhoid fever, or pneumonia, the patient would not live more than a few days. It is not the question of albumen, it is the question of casts, showing disorganization of the kidney, which is the most important point. Moreover, with hypertrophy of the heart, there is a chance at any moment of a rupture of a blood-vessel in the brain, and death from cerebral hæmorrhage. There is also a chance of the heart giving way, and dropsy setting in; and besides there is also the chance of the pericarditis becoming acute, and causing death. In every way, therefore, the outlook for the poor fellow is an extremely grave one. The condition of his kidney is very clear. It first started with an acute nephritis; from that it went on through a subacute into a chronic form, and he has now undoubtedly a large white kidney. Whether he will live long enough for it to contract and become small or not is a question. Rarely does a large one become small, because the patients do not live long enough.

4. This man, W. M., was admitted with extreme pallor of the conjunctiva and skin, which, however, was masked in the face by the sallow complexion which he has. He has extremely pigmented nipples, but there is no pigment elsewhere, neither in the axilla nor on the mucous surfaces. The great point of interest with reference to his case is that he is a gas stoker. The ordinary anæmic symptoms are present, but he has no other physical signs. He has hæmic murmurs both in the neck and heart, but beyond his pallor there is nothing else the matter with him. As an anæmia this case

must be placed in the class of so-called idiopathic, or essential, or pernicious anæmia.

On admission, examination of his blood showed only 20 per cent. of hæmoglobin, 24 per cent. of red blood corpuscles, and one white corpuscle to every 24 of red. The red discs were various in shape and size, and very much paler than they should be. He had been an out-patient for some months before admission, and in July, 1893, there was found to be a large increase in the white corpuscles.

For a few days after admission he had gastric symptoms, vomiting, and constipation, but as soon as these subsided he was put on arsenic, first as liquor arsenicalis, three minims; but this made him sick. He was a day or two after put on one-fortieth grain of arsenic in pill, with two grains of reduced iron, and this dose from twice a day has been steadily increased till he is now taking it five times a day, and is taking it well, and steadily improving under it. He complains of nothing now but that his breathing is still short. The hæmoglobin is estimated at 43 per cent., and the red corpuscles at 40 per cent., showing a marked improvement during the two months he has been in the hospital. The red corpuscles are now much more like normal, running together in rouleaux, and not nearly so pale as they were. The white corpuscles are obviously less in proportion to the red, indeed practically normal. The corpuscles do not stain with eosine. He has greatly improved during the summer, and is now (November) at work, but not at his former employment.

The points of interest in this case are, first, the occupation of the man, and the question whether anything deleterious in his work, such as sulphur, or its compounds, may have destroyed his red corpuscles, as I am inclined to think. There is no malaria, for he has never lived out of Woolwich, and he is living high and well above the river. Then there is no doubt as to his recovery, which removes it from the category of pernicious anæmia, because true pernicious anæmia practically pre-supposes the death of the patient; but there is of course the rarity of such an anæmic condition in a man of his age, except as pernicious anæmia, to be considered. He has no enlargement of the spleen or glands; and there is no deposit of pigment anywhere except just in the one situation, the nipples, which, he says, has existed all his life. If his symptoms are not caused by his occupation, why should he suffer from a curable anæmia?

A LECTURE

ON

EAR DISEASE IN INFANCY AND CHILDHOOD.

Delivered in connection with the London Post-Graduate Course at the London Throat Hospital, July 2, 1894,

By EDWARD WOAKES, M.D. Lond.,

Surgeon to the Hospital, and Senior Aural Surgeon to the London Hospital.

GENTLEMEN,—The subject on which I propose to speak is one not ordinarily discussed, I think, in text-books dealing with diseases of the ear; it is the occurrence of ear disease in infancy, and the way in which ear disease complicates the affections to which infancy and childhood are liable.

It is on the very threshold of life, in the early months of infancy in fact, that the conditions in question are liable to be met with, and the cause of this occurrence may briefly be described thus: Between the ear and some more or less adjacent organs there exists a large amount of so-called "sympathy," a fact that is responsible for many anomalous and unexpected symptoms which may arise in the progress of ailments not originally implicating the organ of hearing, but which thereafter assume the gravest import, as they may involve the issue of life and death. The field of inquiry thus opened, moreover, possesses interests which appertain to the ear as an organ of special sense, apart from the dangers which may attend its implication in the course of other disease. Thus the loss of hearing caused by destructive processes in the ear, occurring as complications of infantile disease, may produce deaf-mutism,—for a child who becomes deaf, from whatever cause, before it has learned to speak, will be dumb also, producing the pitiable object of an intelligent being deprived of two channels of communication with the outside world. Moreover, very much of the ear disease which occupies our attention in later life has its origin in infancy. Our out-patient departments are filled with subjects who have no history, because a discharge from the ear is commonly overlooked in young children of this class, and it is only when they have undergone sufficient mischief to destroy their hearing apparatus to the extent of causing deafness that these children become the subjects of out-patient treatment; by which time the origin of this ear mischief is lost

in obscurity, the parents being too ignorant, stupid, or careless, to take notice of the early and curable stages of the disease.

It will, I think, help my position to describe a case in point. For this purpose, let us consider the case of a child in whom dentition is progressing. It becomes ill, and its behaviour indicates that it is suffering acute pain in the ear. But how is it possible to localize the pain in the ear, seeing the child cannot speak? Anyone who is accustomed to watch these little patients carefully will scarcely fail to recognize in the troubled face, the resting of the head on the nurse, the thrill of pain which passes across its features, accompanied with piteous cries when its position is changed, especially if this be done suddenly, and, more than all, in the constant raising of the little hand to the side of the head,—anyone, I say, accustomed to watch these symptoms cannot fail to recognize in them the most agonizing of all the early sufferings of childhood,—earache.

The point which I wish to emphasise is that the pain thus experienced is not what we vaguely call neuralgia. It is due to a definite trophic change, an inflammation taking place in the deeper seated tissues of the ear, beginning with the congestion and stretching of an acutely sensitive structure, passing on to exudation and suppuration, and capable of being recognised as such if the proper means are taken for doing so. If these symptoms be recognised sufficiently early they may be removed by a free incision of the swollen gums. (I am supposing the case of a child cutting its teeth.) But it often happens that the pathological changes just alluded to have set in before the practitioner sees the patient. The gums, however, are duly lanced, and very properly so, because reflex irritation is thereby lessened; but to the disappointment of practitioner and parent, the little sufferer is not cured. Then commences the orthodox routine of treatment,— cold to the head, hot baths, mustard plasters, perhaps a calomel purge, followed by enemata. Notwithstanding all this the patient gets worse; convulsions set in; and the child dies. I think I am right in saying that in the ordinary run of these cases, in not one in a hundred is the ear ever thought of as the organ that is the chief seat of the phenomena. Here I wish to impress upon you this fact—largely on account of its typical value—that the ear becomes diseased in the instances under consideration in consequence of

impressions proceeding from the gums in the process of dentition, and that these impressions are conveyed from the latter to the former by the nerve communications existing between these two regions—that is to say, the ear is reflexly implicated during dentition. Further, it is owing to the anatomical location of the ear that the mischief thus produced in it extends to the intracranial contents, thereby inducing meningitis, etc. In this way it will be seen that the natural process of dental evolution may result in the death of its subject, because of what is a mere anatomical accident constituted by the normal relationships existing between the nerves of the ear and those supplying the teeth and gums.

It may happen in the progress of such a case that the symptoms suddenly abate, and the child gets well. A little discharge takes place from the ear, or, the friends say, "an abscess has broken in the child's head during the night," and usually no more is thought about the matter. Perhaps the practitioner who is attending the child does not believe in "abscess in the head," and ignores the circumstance altogether. Should the discharge continue, and his attention be called to it, he is careful to inform the friends of the danger attendant upon checking so useful a derivative! At any rate, the disease is let alone. I hope, Gentlemen, that this reflection is not one which applies to the present class of practitioners. Meanwhile the middle ear is undergoing suppurative inflammation, its delicate textures are breaking down, and soon irreparable mischief has been done. Happy is it for the child if one ear, at least, has escaped, which, fortunately, is often the case; otherwise the child will be deaf, and, as a consequence, dumb also.

This is no fancy portrait drawn from my inner consciousness. It is one that I have repeatedly seen, and I am sure that those who are accustomed to out-patient work will bear out the statement that a certain proportion of the cases brought before the surgeon in the aural department arise under the circumstances I have been attempting to describe. Irreparable mischief is accomplished in the ear in the very early months of a child's life in this way, and very often it goes on almost unknown and certainly unaided by those who have charge of the child. Such cases are seen occasionally in all classes of life; and it is well, I think, that your attention should be called to this source of ear disease.

But children have by no means escaped ear trouble, arising from dentition, after they have safely passed this period. Professor Böke, of Buda-Pesth, has distinctly traced otitis in older children to the presence of carious teeth; and in later life a decayed tooth will indicate its presence by prolonged earache, and will even set up an otorrhœa. Mr. Hilton records such a case in his work on "Rest and Pain," and the recognition of such cases will doubtless be more frequent when the possibility of the association referred to is more generally recognized.

It is a matter of some importance to recall the mechanism by which this complicated series of events can be brought about, and this I shall briefly indicate. It involves the principle of the correlating function of the sympathetic ganglia. With each prævertebral ganglion there are associated two sets of nerves—one afferent, the other efferent. Of the sensory nerves that leave the cranium and the spinal cord, all but one or two that are not particularly important, receive branches from a ganglion. Thus, as the vagus passes the inferior cervical ganglion it receives branches from that ganglion at various points, which enter the sheath of the nerve, and pass along with it to the various organs to which it is distributed. An impression communicated to the final distribution of these sympathetic fasciculi in the tissues of the stomach, e.g., follows an afferent course to the ganglion, where it is shunted, so to speak, to another branch, which is efferent, and goes to an artery—the vertebral artery. The effect of such an impression proceeding from the stomach to the vertebral artery is to produce a dilator effect upon the ultimate branches of this vessel, and the general result of such an impression is roughly indicated in the difference between the normal size of the artery and the abnormal dilated condition produced by that impression. The impression is most acutely felt in the small branches of the artery. It thus comes to pass that in consequence of distant impressions which may be shunted in a sympathetic ganglion you get phenomena occurring in regions very distant from the organ in which they are excited; it is in this sense that we speak of the correlating function of the sympathetic ganglia. Returning to our subject, ear mischief excited by irritation arising in the teeth, one channel of excitation—though there are others equally potent—involves the inferior dental nerve, the otic ganglion, and the carotid artery; a

tympanal branch from the internal carotid going directly to the tympanic membrane. Irritation in a tooth to which this inferior dental nerve is distributed implies also irritation in the vasomotor branch from the otic ganglion accompanying it, and that eventually reaches the tympanal branch of the internal carotid, expressing itself in a dilated condition of this artery. It is a short branch, and being in close communication with so large a trunk as the internal carotid is exactly adapted under conditions of dilatation to admit a largely increased supply of blood to the region of the tympanic membrane. You may often see when examining the ear that the vessels of the drumhead swell up as you watch them, so that the membrane becomes more or less injected in consequence of the simple irritation of the speculum introduced into the external auditory canal. Obviously it does not irritate the dental nerve, but those other branches of the fifth found in the canal; the resulting engorgement being induced by a mechanism analogous to that just described. Persistent irritation then of the dental nerves with their associated vasomotor branches causes reflex vessel dilation in the drum membrane and cavity. When this has existed for a time other changes occur—namely, effusion, transudation of cytoblasts, and the formation of pus; in fact, suppurative inflammation is established.

There now arises the question as to the dangers and complications resulting from this occurrence. There is great liability from the first for inflammation so established in the middle ear of an infant to extend from it to the meninges of the brain. This is largely owing to the fact that the squamo-petrosal suture, which early in life begins to close up and ultimately is practically closed, presents in very young infants a considerable gap, through which a process of dura mater passes into the middle ear, to become continuous with the periosteal lining of this cavity. An inflammation of the middle ear can be thus transmitted almost directly to the meninges of the brain; so that in a very young infant, in whom this suture is still open, a middle ear inflammation is almost equivalent to meningitis. Later in life this closes. Through the same fissure branches of the middle meningeal artery pass, and constitute the arterial supply of the middle ear proper. They communicate with the tympanal branch I have spoken of, so that there is a very free inosculation of all the vessels of the middle ear, and any disturbance of equi-

librium in one set of these vessels is readily communicated to the other.

In treating an infant, the subject of inflammation of the ear, an object of primary importance is to secure for it a comfortable position. This is best done by placing it on a large pillow across the lap of the nurse, who is to be instructed not to rock it, as every movement will aggravate its suffering. It used to be the custom for a nurse to take an infant on her knees, and if restless or fretful to jog it, with the object of soothing it. This jogging was very much equivalent to riding a bicycle of the older type. It produced a series of concussions of the spine, and at length the child was lulled into stupefaction by the jerks rhythmically administered to its nervous system. This process has met with the general opprobrium of all educated persons, and probably is not so much in vogue as it used to be.

The condition of children suffering as I have described, who are lulled to sleep by a series of shocks administered to the spinal cord, must be greatly aggravated thereby. The first point in treatment, then, is to secure a quiet and easy posture for the patient. The room should be moderately darkened; it should be free from noise, and of a cool temperature. The affected ear, which will usually correspond with the side on which the gums are swollen, should be uppermost. The child should be fed with a spoon, as the act of sucking increases the pain. Attention to these minute but necessary details will add greatly to the ease of the sufferer, and in proportion promote recovery.

I will take for granted that the gums are lanced. I do not know whether it is now the fashion to lance the gums of infants who are cutting their teeth, but, I take it, that what was a principle twenty years ago must be a physiological principle now, and if the waves of irritation could proceed from swollen gums twenty years ago, I presume they continue to do so to-day. For my part, I should follow the plan, irrespective of fashion, of releasing such conditions of irritation by freely lancing the gums, with a view of diminishing these waves of irritation.

The next point is to get an accurate examination of the drum membrane itself; and this should be done in every case. We have no right to shirk the examination of the locality of a disease simply because it is very difficult to do. In order to overcome the principal difficulty—the child's restless-

ness—it should be placed under chloroform. Chloroform cannot aggravate the condition; it can only palliate it for the time being. Having got the child perfectly anæsthetized, you can examine the ear. You will then be able to see that the pearly lustre of the membrane has been replaced by redness from the injection of its vessels, and occasionally, if the disease has not advanced so far as to impair the transparency of the membrane, the lining of the tympanic cavity will be seen through the drumhead to have a bright copper-coloured appearance. These appearances should satisfy the observer that a very grave state of things is before him. From what I have said about the continuity of the cavity of the ear with the meninges, you will understand the gravity of the situation. Consequently it will be clear to you that the common symptom of earache is not, under any circumstances, to be viewed as a trifling matter. It is not to be treated with a boiled onion or warm oil; nor is it to be entirely neglected as is too frequently done by those who regulate the therapeutics of the nursery. Speaking from my own experience, I am more and more convinced that many obscure and often fatal illnesses in children, which from their affinities are regarded as acute cases of hydrocephalus, or "brain-fever," if properly examined from the aural point of view, would be found to implicate the ear; and I cannot, therefore, too strongly insist on the importance of such an examination being made whenever brain symptoms arise, and their cause is in any respect obscure.

Having confirmed the diagnosis by an optical examination, the pursuit of the treatment will give you no hesitation whatever. The next step in the treatment is to abstract blood from the ear. This is best effected by the application of a few leeches, one, two, three, or more, according to the age of the patient. A little piece of cotton wool should first be placed in the external meatus to prevent the entrance of blood. The leeches should be applied, if possible, inside the concha where the effect of bleeding is most marked upon the deeper-seated structures. If this is impracticable, they may be placed in front of the ear or on the mastoid process. Whatever may be thought of bleeding generally, there can be no doubt that acute inflammation of the auditory apparatus imperatively demands the abstraction of blood. The space is very limited in which the morbid process takes place, the contiguity of important structures

is very close, so that whatever is to be done to secure relief must be done quickly as well as efficiently—all of which requirements will be met by the application of leeches. After the bleeding has ceased, the plug of cotton wool should be removed, and water, as hot as can be borne, should be gently injected into the external canal at short intervals. This simple application is more soothing than any other. Poultices should be avoided, as they promote suppuration.

In the way of medicines bromide of potassium is useful, dose—two or three grains. Tincture of aconite is also of great service, as it lowers the heart's action, diminishes the temperature, and lessens the sensibility of the peripheral nerves. I have very little experience of phenacetin and antipyrin in children's affections, but from what I know of them, especially phenacetin in the ear affections of older people, I should be disposed to give them a trial.

The question arises whether there is any occurrence which indicates that the child is passing into a serious condition. One such, of grave import, consists in the rolling of the head from side to side. This occurs as well in the ear complications of the exanthemata as in those of teething. Other symptoms that set in are of a convulsive character, the thumbs, for instance, being turned into the palms of the hands, the great toe being tightly adducted, and occasionally startings of the whole body. These symptoms should be noted, though they have a less urgent meaning than attaches to the "rolling of the head," which usually comes on later.

What is the meaning of this rolling of the head? Undoubtedly it points to labyrinthine mischief. It means that the expansion of the auditory nerves in the internal ear has become involved in the disease, to the extent of disturbing the equilibrating apparatus, of which the semi-circular canals form a very important part. Nor should it be forgotten that the fact that this organ of equilibration is seated in the auditory apparatus, adds very largely to the importance and to the interest which attaches to all ear mischief. Here we have occurring in early life a symptom which is equivalent to vertigo in the adult. The question may suggest itself to you, why the disturbance of the equilibrating organ should find expression in this semi-rotatory movement? There is no need to seek far for the answer. The faculty of co-ordinating muscular movements, which is

almost entirely regulated by the semi-circular canals (the most recent researches going to show that the cerebellum has nothing to do with it), and those organs in the ear associated with them —the utricle and saccule—is developed by slow degrees, step by step, with the growth of the child. Its influence extends from above downwards. It is first of all manifested in the muscles which move the head. Those who have watched this muscular control developing in a very young infant, will recognise that it is first exercised over the motions of the head, that while the hands move vaguely to seize an attractive object, the head is turned towards it directly and with precision. The lips of the child are its first prehensile organs, and the earliest efforts at muscular co-ordination are exercised to guide these to the sources which supply the means of subsistence. If a child is to seize the breast it must be able to guide its head to the source of the supply of food. Consequently, the process of co-ordination is first developed in the muscles of the neck. These muscles are therefore the first to be trained to act in concert, and being the only ones that do so in very early life it is reasonable to expect that any disturbance in the organ which regulates this co-ordination, should declare itself most markedly where it is most in force, and produce that objectless, rotatory movement of the head. Its presence indicates that the internal ear has become involved in the inflammatory process. It is either being pressed upon through the membranes, which close the fenestræ on the inner wall of the tympanum, or it is actually invaded by the products of this process. The probability is that it is being pressed upon by the collection of fluid in the middle ear. The same phenomena — disturbances of the equilibrating apparatus—are manifested in the infant, just as they are in adults, from a similar cause; in adults there is vertigo, in infants this rolling of the head from side to side. If, however, you see this phenomenon, you may be certain you are at the final stage of an ear trouble, the probabilities being that you will have no other chance of affording relief to the patient, as in a very short time the meninges of the brain will be involved. There is, however, just a possibility of affording further relief even under these circumstances. If this condition results from the pressure of fluid collected in the middle ear, by puncturing the drum membrane we secure the same effect as the

opening of an abscess,—the symptoms are entirely relieved thereby.

I do not think that in this phase of infantile disease occurring in connection with teething, in which the process is from the commencement acutely inflammatory, rapidly extending to the meninges, that you will often get suppuration. If the ear should suppurate, you will probably have the same process going on in the submeningeal space as well. But at any rate you may give your patient the chance of getting relief by incising the drum membrane. This chance occurs especially in those affections of the ear which complicate the exanthemata. *Implication of the ear organs in the exanthemata* is one of the most frequent causes of fatal issue in these cases. It is generally through the ear that the brain becomes implicated in them. There are one or two points about this implication of the ear in the exanthemata that are worth remembering. One of these is the method of its occurrence. The lining membrane in the middle ear of a child, as in an adult, is continuous with the mucous membrane of the post-nasal space, the nasal cavity, and the fauces, through the Eustachian tube. The rash of an exanthem must of necessity be as likely to occur in the middle ear, as it is in the fauces, the conjunctivæ, the nasal mucous membrane, and the mucous tract generally. The probabilities, the certainties, I might almost say, are that the processes that occur in the ear do so as the result of such a rash taking place there. The seriousness of their occurrence in the region of the Eustachian tube and the adjacent cavities, arises from the consequent tumefaction of the mucous membrane, whereby the tube is closed, and the ear cut off from the cavity into which it should be able to drain. When the drum cavity is inflamed by the occurrence of a rash in it,—scarlet fever, measles, or small pox,—the ordinary outlet for such exudations as would occur, is closed. If the adjacent mucous membrane is already swollen and the tube closed, it comes to pass that pressure is set up, and you may early in an exanthem get those phenomena to which I have alluded in the case of implication of the ear during teething. Then convulsions set in, and if the deeper seated regions are involved, rolling of the head also.

It is when the ear mischief of the exanthem has arrived at this stage that it usually attracts the notice of the surgeon by the occurrence of discharge from the external canal. The destructive processes take place with great rapidity. In twenty-four hours a child's ear may be absolutely destroyed. It is, therefore, of the more importance that in the exanthemata you should be able to get a clear view of the situation, and that you should be able to direct your treatment to the relief of the urgent conditions already established. There must be no hesitation in having recourse to chloroform for the purpose of an examination. Should you find bulging of the drum head it will be your imperative duty to incise that membrane,—not so difficult a matter as it may seem, since in infants the external canal is much shorter than in adults, although, it is true, the membrane itself is on a much more horizontal plane. With ordinary skill you can do no harm. I used to suggest the use of a guarded instrument for this purpose. Some one, in reviewing my suggestion, said it was an unsafe thing to give an ordinary practitioner an instrument with which he felt entitled to "jab away" at the ear. I do not suppose any surgeon would do so; but it may be some sort of comfort to feel that you are not so likely to do harm by making use of a guarded blade till you have got down to the desired spot. Having reached this point press the spring, and give the instrument a slight upward movement to ensure a sufficiently free incision. Although many years ago I used this instrument I should not do so now, preferring the ordinary knife in vogue for incising the drum head. However, this instrument has done duty in its day, and may do so again.

As to the subsequent treatment, it is very important in all these cases to get the nose and the post-nasal space, as well as the external auditory canal, well washed out and cleansed from the secretions that accumulate therein. The ordinary alkaline lotion is probably the best for this purpose, consisting of—bicarbonate of soda, half an ounce; carbolic acid, half a drachm, to a pint of water. With this alkaline lotion, warmed by diluting with hot water, the nose and external ear should be syringed very gently three or four times a day. This alone will tend to wash away the obstructions in the Eustachian tube, and allow the pus that accumulates in the middle ear to escape *per vias naturales*. Any other treatment you may fall back upon will of course be guided by your judgment. The important point, however, is to allow a free escape, both externally and through the Eustachian tube, of anything like a collection

of purulent and decomposing fluid in the middle ear. It will assist this object to make occasional use of Politzer's process of inflating the middle ear, especially when discharge from the external ear has been established: it is essential to observe great gentleness in its use, and may perhaps be better avoided altogether during the acute stages of the illnesses thus briefly described.

CLINICAL NOTES.

WITH MR. ALBERT CARLESS' OUT-PATIENTS AT KING'S COLLEGE HOSPITAL.

A Case of Chronic Osteitis of the Tibia in a man aged about 55 years.

This patient was first seen one year and eight months ago. He then complained of intense pain in the tibia. The pain was typically "bony," that is, it was of a deep boring nature, and worse at night. The tibia was enlarged in all directions, lengthened to the extent of one inch, and presented a distinct antero-posterior curve.

Its appearance suggested at first sight a case of osteitis deformans; but there were no other signs of this condition; thus the femora were not curved, the spine was not kyphotic, the calvarium was not enlarging, and he did not present the bent head, elevated shoulders, and waddling gait characteristic of the latter disease. It is true that this complaint may affect one bone alone, and then usually the tibia, but on the whole one was inclined to consider the case one of chronic osteo-periostitis, possibly associated in origin with osteo-arthritis.

The patient denied syphilis, and gave no definite rheumatic history. Linear osteotomy, as described in text-books, and often performed, i.e., by making a linear incision into the bone by a Hey's saw, does not usually prove sufficient in these chronic cases, and therefore, the following operative measures were adopted. An incision was made down to the bone, six inches in length, the periosteum was stripped back, and, corresponding to this, a canal was chiselled and gouged out of the exposed bone, laying open the medullary cavity, thus making "a hole to let the pain out," as it was aptly termed

by a former patient who had gained relief by a similar operation. No pathological condition was met with in the interior of the bone. The superficial soft structures were now loosely drawn together, covered with purified protective, and dressed in the ordinary way. By this means the canal filled with blood clot, which by its subsequent organization repaired the breach in the bone. This method of healing is to be preferred to that of packing the hole in the bone with gauze, in that it is more rapid, and necessitates less interference with the wound during the after-treatment. The opposite tibia is also slightly enlarged, but this has been improving under treatment with iodide of potassium. Both ankle-joints are also somewhat swollen.

Several cases of Gonorrhœa were then seen, and

The use of injections in the treatment of Gonorrhœa discussed.

Considerable discretion is needed in recommending injections for this disease, for not only are they often quite unnecessary, but in many cases they do actual harm. In the early stages they should never be employed, but in the free-secretion stage, *most* cases can be cured by simply administering the oleo-balsams, keeping the patient's bowels open, and forbidding him any alcohol. In neglected and chronic cases, injections are often most valuable, and amongst those to be recommended are a three per cent. watery solution of ichthyol, or a solution of permanganate of zinc, $\frac{1}{4}$ gr. or more to the ounce. One of the best consists of:—

R	Sulphate of zinc ...	...	...	gr.iij
	Tincture of catechu	...	...	ℳx
	Tincture of opium	...	...	ℳv
	Glycerine	...	...	...	ℳxv
	Water ...	...	...	ad ℥j

In using injections, it is essential that the urethra is first washed out and cleansed from any discharge lying in it. To accomplish this, the natural act of micturition suffices, i.e., the injection should always be employed after voiding urine. The nozzle of the syringe is then inserted into the urethra, and the lips of the meatus closed over it by the fingers; about half an ounce or more of the fluid is introduced into the urethra, and should be retained here for about one minute, being subsequently allowed to flow away.

Too early adoption of injections may light up

epididymitis, and I personally incline to the old idea that the latter complaint is more common in cases treated by injections than when they have not been employed.

A case of Chronic Enlargement of the lower ends of the Tibia and Fibula.

Here, the lower ends of the bones were expanded in every direction, the breadth of the internal malleolus being two and a half inches, and the external malleolus was similarly enlarged, the whole presenting a collar-like appearance around the ankle ; the foot, however, moving perfectly freely, and without pain under this mass. There is, however, a good deal of pain in the bones— worse after exercise and at night. The masses of bone somewhat simulated the appearance of callus, but there is no history of injury to be obtained. A syphilitic history was given—the primary infection dating many years back.

The diagnosis of this case, which is a somewhat obscure one, rests between (a) chronic osteitis of the lower ends of the two bones, secondary possibly to some deep central affection of syphilitic or tubercular origin, not implicating the joint, but probably causing osseous ankylosis between the two bones ; (b) syphilitic disease of the joint, which is often painless, and leading on to a similar affection of the bones, the joint trouble in the meanwhile having cleared up ; (c) osteo-arthritis, mainly affecting the lower end of the bones, and causing but little interference with the articular tissues ; or (d) a somewhat unusual form of Charcot's disease, in which the effusion into the joint is minimal. Probably the last is the correct diagnosis, since other evidences of tabes are also present in the shape of fulgurating pains down the limb ("like a flash of lightning," the patient states), loss of patellar reflexes, and the Argyle-Robertson pupil.

The treatment adopted has been the administration of large doses of iodide of potassium and strapping with emplastrum ammoniaci cum hydrargyro.

The patient thinks the ankle is better, as the pain is less, and the swelling distinctly smaller.

Some cases under treatment for acquired syphilis having been seen, the question arose as to

The duration of treatment of syphilitis.

One needs to use great care in giving a " clean bill of health " to a patient who has been under treatment for syphilis; for cases differ widely in their reaction to mercury, and in the length of time needed to remove the symptoms. Speaking generally, the earlier the disease is diagnosed and anti-syphilitic treatment adopted, the shorter will be the duration of treatment ; but in all cases one likes to keep patients under treatment for at least eight months, giving two grains of pulv. hydrarg. cum crêta three times a day for about five months, and afterwards one grain three times a day for three months.

If, however, the disease is not recognised for eight or more weeks after infection, a longer course, at least one year, of treatment is necessary, for the disease has obtained a firmer hold of the patient and is, therefore, more difficult to eradicate. This is especially true of cases of extra-genital syphilis, which are frequently only diagnosed when the secondary rash has appeared.

The question as to how long a time should elapse before marriage can be contracted often arises, and it is always an extremely anxious point to be called on to give a decision. The rule should be, that marriage must never be entered upon until at least one year after *any* sign of syphilis has been manifested ; and even then it will be wiser to give the patient a mild course of mercurial treatment for about three months before marriage in order to minimise the risk to the woman.

A Case of Tinea Tonsurans (Ring-worm) was then shown :—

The patient, a boy of 12, on examination, presented an almost bald condition of the posterior two thirds of the scalp, the hairs over this region being broken off, short, "stubbly," and twisted. There was general scurfiness over the same area. Over the forehead, cheeks and back of the neck were several round patches, scaly on their surfaces, with an erythematous and thickened margin, which were evidently due to the same condition.

Treatment.—As the disease is parasitic in nature, antiparasitic remedies are adopted.

On the skin, tincture of iodine painted on gives good results, as here the tricophyton tonsurans is not deeply seated in the follicles.

But on hairy surfaces, where the parasite pierces the hair follicles, and invades the hairs, treatment is much more difficult. For some time back I

have adopted the method of treatment suggested by Professor Unna, of Hamburg, and with excellent results.

This treatment consists, first, in cutting the hair quite short over the greater part of the scalp, and the application subsequently of two ointments.

No. 1 consists of:—

R	Ac. salicylic	...	...	gr.x
	Ac. chrysophanic		...	gr.xxv
	Ichthyol ...	...	...	30 per cent.
	Vaseline ...	...	ad	ʒj

No. 2 consists of:—

| R | Unguentum ichthyol | ... | 20 per cent. |

The head must be washed every morning with soft soap and water, and for the first four days of the week, No. 1 must be applied to the scalp, spread freely on linen, the whole covered by a piece of gutta percha tissue, and secured by strapping, or by a small cloth skull cap.

On the fifth morning the second ointment should be applied and continued with similar precautions to the above, until the end of the week, when the same cycle of treatment is renewed.

As a rule cases are cured thus in about three weeks, but neglected cases may take six. A careful microscopic examination of the hairs is needful before a case can be pronounced cured. It is worthy of note that adults very rarely acquire ringworm.

A baby was then shown with a Nævus on its Back.

In this position since cosmetic effect is unimportant, excision is the best procedure. This should be performed by the use of semilunar incisions. The subsequent cicatrix is linear; and if healing by first intention is obtained, the cure is correspondingly rapid.

Strangulation should never be employed in the treatment of nævi, since wherever this is possible, excision can be performed. Moreover, a much larger scar is left, and as the nævus has to slough away, and the wound therefore heals by granulation, a favourable result is much more slowly attained.

When the nævus is in places where excision is impossible or contra-indicated, as, for instance, on the tongue, face, or lips, then two methods of treatment are open: *injection*, or *electrolysis*.

If the circulation behind the nævus can be controlled, then *injection* may be adopted. A hypo-

dermic needle should be introduced, and minute drops of the coagulant in the syringe scattered about here and there throughout the tumour. Either tinct. ferri perchlor., or liquid carbolic acid, is employed for this purpose. If the latter is used, the greatest care must be taken to prevent it from coming in contact with the under-surface of the skin. The control of circulation may be attained by temporary strangulation of the base of the tumour by means of sutures tied round hare-lip pins passed under the nævus, or other methods. It must be maintained for five to ten minutes after the injection. The tumour subsequently becomes firm and hard from thrombosis, and gradually shrivels away by the formation of fibro-cicatricial tissue and its subsequent contraction.

Where the circulation cannot be controlled, or where it is desirable to leave a very minute scar, *electrolysis* gives the best results. The positive terminal is connected with one or more needles, and these are plunged into the mass, while the negative pole is attached to an electrode, and placed on some suitable part of the surface of the body. From the positive pole, acid compounds are liberated, and if the needles are of steel, iron salts are produced which assist in determining thrombosis. From the negative pole, if inserted into the tissues, caustic alkaline compounds are set free which cause sloughing of the tissues, and a loose spongy coagulum alone forms. For this reason the positive pole should be alone employed.

The current used is of the strength of 200 milliampères; if, however, both needles are inserted into the nævus, half that current suffices. The tumour is felt at the time to become harder, and subsequently shrivels and shrinks. More than one sitting is often needed to effect a cure.

Tattooing of the skin by the iron salts may result, and hence needles with a coating of shellac should be employed. It must be remembered, however, that they must not be soaked in carbolic acid solution for any length of time before use, or the coating will be found to peel off.

A case of Osteo-arthritis of the Knee in a young girl,

with effusion into the joint, and proliferation of the villi of the synovial fringes.

On examining the knee, there is evidently effusion into the joint, and on movement, soft, not bony, crepitus is obtained, not between the patella

and the articular surface of the femur, but in the interspace between the bones themselves; and on careful palpation the hypertrophic fringes of the synovial membrane can be felt moving under the fingers. In young persons as a rule, both bone destruction and new bone formation are slight in amount in comparison with cases occurring in older people, and this patient is no exception to the rule. I wish also to draw your attention to the fact, that although there is, as a rule, but slight effusion in cases of osteo-arthritis, yet instances occur in which it may be abundant, and in such overgrowth of the synovial fringes is generally a marked feature. Many cases of so-called "hydrops articuli" are of this nature.

In young persons where the disease is limited to one joint, good results are often gained by excision.

In this case Heberden's nodosities are present at the ends of the phalanges, and the inter-phalangeal joints are somewhat enlarged, but there is no ulnar adduction of the fingers, although this latter is a common feature of the disease when affecting the hand. This would seem to suggest a general origin of the disease, and to contra-indicate operative treatment.

The treatment adopted has been to strap the knee-joint and to keep it at rest, whilst a mixture containing citrate and iodide of potash has been administered internally with advantage.

REVIEW.

Annual of the Universal Medical Sciences. 5 vols. Edited by CHARLES E. SAJOUS, M.D. (F. Rebman, London).

Published at £3 5s.

Nothing can give one such an idea of the enormous activity of the progression of medicine as an attempt to review a work of this description. This is the seventh year of the production of the work, and we can say that its reputation is fully maintained for completeness of reference and suitability of selection. In the space at our disposal we can give little else than a very brief *resumé* of the means of its production and the results attained.

Dr. Sajous is at the head of a small army of editors; the actual associate editors number seventy, while the "corresponding editors, collaborators, and correspondents" amount to over two

hundred. Such a subdivision of the work enables it to be done in a most complete manner, each editor being responsible only for a small section of which he is a complete master. No less than 1,153 periodicals are brought under contribution, and the grain of them is all carefully sifted from the chaff week by week or month by month; besides these, 178 monographs, theses, and books of a less ephemeral nature than periodicals, have been ransacked for information. This is the method by which the "Annual" has been produced. The result is what we should expect from such a huge engine of production.

Vol. I. contains the pith of all that has been written during the year on the diseases of heart, lungs, pleura, intestines and their glands, on parasites, on zymotic diseases, and eruptive fevers, on the urinary organs, rheumatism, gout, and the blood.

Vol. II. Nervous diseases, organs of generation, pregnancy and parturition, puerperal diseases, and diseases of new-born infants and children.

Vol. III. Surgical affections of every region—from those of the nervous system to ordinary fractures, and dislocations, and operations for, and discussions on the nature of tumours, etc.

Vol. IV. is devoted to what are usually termed specialities; *i.e.*, diseases of the eye, ear, throat, skin, etc.; legal medicine, demography, and bacteriology.

Vol. V. includes a digest of all progress in therapeutical measures, using the term in its widest sense, including electricity, baths, etc., besides drugs.

An index of sixty pages concludes the work; this is divided into three columns: general index, therapeutical index, and a column of authors quoted; this is in addition to an index at the end of each volume.

It is impossible to deal more at length with a work of this size. As a book of reference we can warmly recommend it for its completeness and the ease with which any given subject can be hunted up, but owing to its price and size we doubt whether it can be a commercial success. The cost of production must be enormous, and its sale must be limited, unless indeed many men with but scanty leisure should choose to leave all current literature severely alone and study this epitome.

None the less, we cordially congratulate M. Sajous and his collaborators on the success of their gigantic enterprise.

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

HOOPING COUGH.

Delivered at Charing Cross Hospital, Oct. 27, 1894, by

JOHN ABERCROMBIE,

M.D. Cantab, F.R.C.P. Lond.,

Physician to the Hospital.

GENTLEMEN,—The case in the Victoria Ward of a little girl with hooping cough has afforded you abundant opportunities of studying the characters of a typical case of this disease, and I assume that you are now familiar with the characteristic paroxysm of the malady.

It is usual in descriptions of this disease to divide the symptoms into three stages, viz., the catarrhal, the spasmodic, and the period of convalescence, but this is purely an artificial division which can hardly be maintained clinically, and is of no real advantage. It is true that in the early period the cough may present none of the features which we recognize in the disease, for the patient may simply appear to be suffering from an ordinary catarrh, and it may be ten days or so before the characteristic paroxysms appear. On the other hand, in some cases the patients hoop from the beginning, although in most cases the cough is at first apparently simple, and only gradually becomes paroxysmal towards the end of the first week. The typical fit of hooping cough is very characteristic, and consists of a series of violent expiratory coughs, followed by a noisy inspiration; this is generally followed by a recurrence of the fits of coughing, terminating in another and more prolonged and more noisy inspiratory hoop.

The child (for the patient is generally a child) may now bring up some viscid mucus, and is not infrequently sick, but in severe cases a third bout of coughing and hooping will immediately follow before the expulsion of the mucus or the emptying of the contents of the stomach.

The attack is often preceded by a state of nervous agitation, and the child, always being aware of its approach, stops playing with its toys, while it clutches hold of something for support.

If the attack be severe, the child will be much exhausted, and will lie quiet for a time after it is over; but if it is not so severe the child may immediately take up its toys again, as if nothing had happened. The number of paroxysms is very variable, but if there were, on an average, one an hour, we should be dealing with a case of a severe character.

As regards the physical signs, there may be none, or there may be a pulmonary catarrh, and in more severe cases some evidence of areas of collapse in one or both lungs. Fever is not necessarily present, but there may be slight fever during the second stage, and especially if there is any lung complication. In all cases of cough in children, it is well to make certain as to whether there are any other children ill in the family, or in the same house with cough. Amongst the symptoms which may suggest the true nature of the case in the early stages, is a cough which will be particularly hard and irritable, especially at night, and there may also be some coryza and suffusion of the eyes. If the child were puffy under the eyes, or had epistaxis, it would be very strongly suggestive of the true nature of the case. The hooping is not absolutely essential; babies under six months old do not, as a rule, hoop; they have paroxysms of coughing and turn red, or sometimes more or less black in the face. In adults it is common that there should be no hoop, and in cases where grave lung complication is present, the hoop is often absent. In consequence of the severity of the paroxysms, we may get production of hernia or hæmorrhage from the nose, mouth, lungs, ears, or into the conjunctivæ; this latter is extremely characteristic, as in absence of bruising round the eye, the only possible other cause would be an operation for strabismus. Hæmorrhages into the skin are very rare.

The chief difficulty in diagnosis is in the early stages, and sometimes a correct diagnosis is impossible then. The main thing we should have to exclude would be acute tuberculosis; in both we might have cough, and signs of general pulmonary catarrh, cyanosis, and fever. In tuberculosis the child would probably be more ill than could be explained by the physical signs, whilst in hooping

cough it is just the reverse, the child does not seem so ill as the physical signs would lead us to expect.

The duration is very variable. It may last from one to two months or more. The child may be allowed to mix with others two weeks after all the hooping is over, provided at least six weeks have elapsed since the onset of the illness. Cases are infectious from the commencement of the cough, but how long the infection may last is not known. The child may go on hooping for four months or longer, though it is probable that after this length of time the infectiousness is over, and the peculiar cough is simply the result of habit. But a child who has had hooping cough will be very likely to hoop again, if it should get another cough within a few months of recovery.

Second attacks are probably very rare; I have never seen two attacks in any child myself, but on several occasions I have seen hooping cough (which proved fatal) in children said to have had the disease before.

Amongst complications, the most important are those referable either to the lung, or to the brain. Collapse of the lungs, or of part of the lungs is very common, because during the paroxysm of coughing, the lung is emptied of air to an unwonted degree, whilst during the laboured inspiration through the narrowed glottis, the viscid secretion of mucus in the tubes opposes a difficulty to its efficient entrance, and so collapse results. Acute emphysema is common, even in the early stages, and some degree of bronchitis is almost the rule, and can hardly be considered a complication. Catarrhal pneumonia is decidedly formidable, pleurisy and empyema are less common, and hæmorrhages into the substance of the lung are occasionally met with. Convulsions may be due to asphyxia, or to congestion, or to hæmorrhage into the brain or meninges, or to thrombosis of the sinuses. In a rickety child convulsions are common, and also in young children with grave lung mischief.

The vomiting is not often so serious as to cause anxiety, though it may lead to wasting, and some degree of looseness of the bowels is rather common. Epistaxis and hæmoptysis, if copious, might give rise to trouble. Sub-lingual ulceration has had more attention paid to it than it deserves; this is due simply to the scraping of the under surface of the tongue over the lower incisor teeth during the paroxysmal cough, and therefore never is met with in babies who have not cut the lower incisors.

In scrofulous children we are apt to get large caseous glands left behind, and acute tuberculosis as an ultimate sequel. Epilepsy, hemiplegia, and diffuse sclerosis of the brain have all appeared to date from an attack of hooping cough, and I recollect one very interesting case of septicæmia where a child died from meningitis, with pleurisy on one side, and empyema on the other, in whom I found a decomposing blood-clot in each internal ear, which had evidently been the cause of the septicæmia, and had resulted from hæmorrhage during a paroxysm of coughing.

As regards the prognosis, there is no doubt that a considerable number of children die every year of this malady. In a child over two years of age, if the case is uncomplicated, you may give a favourable opinion, the younger the child the more grave is the prognosis, and in infants under six months it is exceedingly bad; every complication increases the danger. In rickety children under two years of age the prognosis is always grave, there being great danger of lung trouble, and of brain mischief. Complications usually come on in the first ten days; after that period has elapsed, without any complication supervening, the prognosis would become favourable, even in a young child.

With respect to the pathology—there are no post-mortem appearances peculiar to the disease, though enlargement of the tracheal and bronchial glands is very common, as is also hyperæmia, and catarrh of the mucous membrane of the trachea, larynx, glottis, and posterior nares.

No specific bacillus has as yet been found, the disease is highly infectious, and as a rule all who are exposed take it. Very short contact is necessary, and it may be carried by a third person in the clothes. In a considerable proportion of cases the attack follows immediately upon measles, but hitherto the explanation of this fact completely escapes us.

In the slightest cases no special treatment is necessary except seclusion from other children; if the weather is mild such a patient need not be kept indoors. In all cases attention should be paid to the diet and bowels; and when possible, the child should be placed in a large and well-ventilated room. In the early stages, expectorants and diaphoretics may be given, if there is much cough. When the paroxysms supervene we may add some tincture of belladonna, and bromide of ammonium to the mixture. You may give two

minims of the former, and a grain of the latter, every three hours, to a child of a year old. Some prefer to give liq. atropinæ sulph. in place of the belladonna, in half drop doses. Others recommend tinct. camph. co. instead; and no doubt this is useful in the later stages. In some cases, I have given chloral hydrate in half grain doses, but an infant taking any of these remedies in the doses mentioned, ought to be under very constant supervision. Another line of treatment much recommended, is to brush out the larynx with solutions of nitrate of silver, cocaine, or resorcin. The manipulative difficulties have deterred me from attempting this method of treatment hitherto, but I have often thought that a vapour of carbolic acid produced by placing some in an ordinary kettle on the fire, or in a special steam kettle, was beneficial. Complications when present, should be treated on general principles; in the later stages, and in cases which tend to be stationary, I have always thought that cod liver oil does more good than anything else; and a change of air whenever it can be secured should be resorted to.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

AMONGST the cases seen on October 29th were the following:

Multiple Sarcomata in Connection with Bones.

A man, aged 62, who had been sent from the London Hospital by Mr. J. Hutchinson, Jr. He was the subject of four hard and apparently osseous tumours developed in connection with bones. One was on the sternum, one on the right scapula, and two on the ribs of the right side. The largest, which was as big as a fist, was on one of the ribs, and it had been the first to appear, the patient dating it ten months ago, whilst the others had followed not long afterwards. The tumours had been comparatively painless, and they had not as yet in any way interfered with the patient's health. There were no indications of any internal growth, which could have served as a primary source of infection. There was no history of syphilis.

Comments.—Cases of primary sarcoma of bones are rare in senile periods, and multiple ones yet more so. These are remarkable on account of their extreme hardness, although they have grown rather rapidly. It is to be noted that they are all in connection with flat bones, and all to the right of the median line.

Case Illustrating the Effects of Inherited Syphilis in Adult Life.

The subject of this case was a woman, aged 37, who had been under my observation when a child, twenty-four years ago. She then passed through a very severe attack of interstitial keratitis, and had characteristically notched teeth. She is now a tall, well-grown woman, her features are not malformed, and by wearing down of the teeth all traces of the notches have been lost. Thus the recognition of taint at the present time would be very difficult had we not the history. She is, however, the subject of extreme myosis, her corneæ show the steel grey haze, which is not unfrequently left by keratitis, and, what is of more importance, she is absolutely deaf. Her hearing was lost in 1871, the deafness having become complete on both sides within a fortnight of the first ear symptoms. She can now be communicated with only by signs. In her corneæ, near to their borders, there are some white lines and patches, and in the upper part of each there is a distinct white arcus. Her sight is very defective, and we are told that not unfrequently, on a sudden movement of the head, she is in total darkness for a few moments. These conditions indicate atrophy of the optic nerve and great narrowing of the arteria centralis. With the patient comes a younger sister. She is only 18 months younger, but she has good teeth, and has never shown a single trace of inherited taint.

Comments.—In this patient we have a good example of the terrible results of inherited syphilis as regards the organs of special sense. The patient is absolutely deaf and almost blind. Yet you have seen that she is well-grown, and that, were it not for my knowledge of her as a child, it would be very difficult to establish a diagnosis of inherited disease. The form of deafness from which she has suffered is, however, in my opinion, in itself conclusive. We know of no other form of ear disease in which, without any obvious destruction of parts, complete and permanent deafness in both ears can be brought about in the course of a

few weeks. Yet this, unfortunately, is not by any means a very rare occurrence in the subjects of inherited syphilis; and the statistics of the aural departments of our hospitals show that inherited taint is the cause of complete deafness in early life in a very large proportion of cases in which it is met with. Our patient further illustrates a remark which I have often had occasion to make, that the subjects of inherited syphilis in adult life are, with the exception of the risk to the organs of special sense, not prone to suffer from any particular form of disease. They do not become the subjects of lupus or any other skin affection. The two sisters, whom you have seen, are very much alike, one as well developed as the other; almost the only difference is that one of them is deaf and blind.

Case of Alopecia Areata in Early Childhood.

The subject of this case is a boy aged 8, brought by Dr. Bullock. He was the subject of characteristic alopecia areata. The history is that five years ago, when 3 years old, the hair began to come off in patches the size of shillings. These patches occurred first on the occiput, but subsequently on the temples. It was not known that he had ever been exposed to the contagion of ringworm, and the patches were from the first quite smooth. According to his mother's account, the hair grew again on the temples, and was kept for a time, and two years later, under the use of an ointment it grew well over the whole head. No further loss of hair occurred until January, 1894, when the hair again began to fall in round patches. At present, under the use of chrysarobin ointment, there are again signs of its growth. The boy is in good health, and has not suffered from headaches.

Comments.—You will see that we have in this patient a typical example of alopecia areata, exactly as we meet with it so frequently in young adults. The remarkable point is that the patient is a child. It is very unusual indeed to encounter such a case beginning at the age of 2. My creed has long been that this malady is cryptogamic, and that the cryptogram is the same as that of ringworm and tinea versicolor, although of course in a modified form of growth. All the clinical evidence with which I am acquainted, tends to the belief that these maladies are transmutable. My suspicion is then, in such a case as the present, that the patient

has probably obtained his malady by direct contagion from another case of alopecia. That there is any neurotic element in this disease, or that it stands in any sort of connection with the general health I wholly disbelieve, and this case, I think, supports my scepticism.

Alopecia Areata in a Young Boy proceeding to Complete Baldness.

A boy named Inskip, aged 12, brought by Dr. Fletcher, of Camden Road, was an example of complete alopecia at an unusually early age. His baldness was absolute so far as the scalp was concerned. His eyebrows were also lost, and his eyelashes were very small. The history given was that at the age of 3 years the hair began to fall in round patches, and that in the course of a year the baldness was complete. From that time there had never been any growth of hair. There was no history of previous ringworm, the mother's statement being definite that the patches from the first were smooth and glossy.

Comments.—Alopecia areata at this early age is rare; but the case is by no means unique. It would appear that the disease developed just as it does in older patients, by the formation of round patches, which spread at their edges. We have, therefore, no reason to separate the case from those in which alopecia areata is seen later in life. As to cause, I should suspect direct contagion from some patient who had the same disease, and not from ringworm. This, however, is only conjecture. That it is not a very actively contagious malady is proved by the fact that, although the boy has lived, since the affection began, for nine years, in a family of eight brothers and sisters, none of the others have taken it.

Infective Lymph-Angioma (Lupus Lymphaticus).

The patient, a girl of 12, had on the right side of her chest, just below the border of the axilla, an oval patch about 1½ inches long, which consisted in part of clustered transparent vesicles and in part of vesicles of a dusky plum tint. Under this patch and placed subcutaneously was a doughy-feeling, ill-defined swelling, no doubt a nævus. There was, however, no trace of a congenital nævus in the skin itself. Near to the principal patch there were a number of scattered

satellite vesicles, some of them transparent and others containing blood.

Comments.—It is not very often that one has an opportunity of showing side by side with the patient a drawing of disease so exactly representing the conditions as the one which I now produce. In the drawing which I show, the patch is placed on just the same part—the side of the chest of a young lady. The vesicles are exactly like those in our patient, and just above the group is a white scar, left from an operation by Sir William MacCormac for cure of a nævus. At the time that the nævus was treated there were none of the vesicles of the skin, which at the time the portrait was taken, some years later, were so conspicuous and extensive. We have therefore conclusive proof that the disease was infective and aggressive. Precisely the same statement is true of the patient now before us, and of all other examples of the malady which I have seen. It is not a quiescent lymphangioma, but an infective inflammation of the skin, attended by the development of lymph vesicles. Its groups spread at their edges, and produce satellites. It is, moreover, very liable to attacks of a sort of erysipelatous inflammation. On account of these characters, I felt justified many years ago in describing this malady as one of the lupus family, a lupus lymphaticus, and that such is its natural position I still hold. It is a lymphangioma without doubt, but this word recognises its pathological anatomy only and not its clinical features; the latter are those of a lupus. The case, which Dr. Sequeira has been good enough to bring us to-day, is exactly like all the rest. In a majority of cases the changes begin in proximity to a congenital nævus; but I do not think that they are ever seen during the first two or three years of life, and in some there is no history of any nævus whatever. As we all know, it is no part of the clinical history of nævus that it should spread infectively. Some of the cases appear to have been influenced or located by external irritation. Thus, in two boys of whom I have portraits, it had apparently been produced by the rubbing of the braces, and in our present case, as well as in the portrait just shown, its occurrence near the border of the armpit in girls is suggestive of suspicion that the part may have been rubbed by the dress.

As regards treatment, it must be precisely that of lupus. You have to deal with an infective form of dermatitis, and you must completely destroy the infective elements if you would cure the disease. This may be done either with nitric acid or with Paquelin's cautery, but it must be done effectually and in all probability it will need repetition. If once completely destroyed and a healthy scar produced, then, as in a patch of lupus, cure will be permanent.

Question of Diagnosis between Congenital and Acquired Syphilis.

The subject of this case was a married woman, aged 23, who was suffering from an extensive lupoid ulceration of her face and lips. Her tongue was lumpy and thickened, and her whole mouth sore. She was the subject also of phthisis, and her father was said to have suffered from lupus. There were, however, features in her case which, apart from the condition of her tongue, made it quite certain that she was the subject of syphilitic taint. Both tibiæ were enlarged and bulged forward by osseous nodes, and her legs were covered with large scars. All of them, however, were soundly healed.

Comments.—We have in the patient before us a very interesting problem. First, as regards the lupoid ulceration of her face and lips, it is impossible to say whether it is the result of syphilis or struma, or whether we should regard it as a hybrid between the two. There is little or no doubt that the patient is tuberculous; but there is absolutely none that she is syphilitic. The condition of her tongue is such that we do not see in scrofula, and, although the large scars on her legs might be taken for the relics of Bazin's malady, they are far more like those of syphilitic ulcers. The nodes on her shins, however, clinch the diagnosis. There is no doubt whatever that they are the result of specific disease, and, from the amount of deformity of bone which has been produced, I should esteem it almost certain that the periostitis occurred during early life, before the bones had attained their growth. We never see tibiæ so bent, thickened and deformed in those who have acquired syphilis after adult age. Thus it is seen that we have to raise the question as to whether she is the subject of inherited taint. I am inclined to the opinion that it is a case in which syphilis must have been accidentally contracted in infancy, and that it was not actually inherited. For this opinion I will state my reasons, premising that we cannot get from the girl herself any history

which helps us. It is exceedingly uncommon to find any form of skin disease occurring in connection with inherited syphilis after the period of infancy. In infancy, of course, rashes are the rule; but they disappear in the course of a few months and after that, with the very rarest exceptions, we never encounter either the rupial or lupoid eruptions which are so common in the later stages of acquired disease. Now this patient has had very severe skin disease of two different forms at a comparatively late period. We may next note that disease of the tongue, sclerosis, such as this patient shows, is scarcely ever seen in connection with inherited taint. I feel, therefore, almost sure that this patient must have had the acquired form. It is of course possible that she may have had an inherited taint also. There is, however, no proof that she has had interstitial keratitis, and as her teeth have fallen out we cannot avail ourselves of them for diagnosis. We have in the museum only one or two drawings which illustrate the occurrence of severe skin disease at late periods in the subjects of infantile syphilis. One of these I now show you. The child was under my care in the London Hospital many years ago, and was, as you see, covered with a sort of rupial lupus, very like that of the patient who has just been before us. In the same ward lay the mother of the child, herself the subject of tertiary syphilis. When the portrait was taken I believed that the child was the subject of inherited taint; but subsequently facts came to my knowledge which made it probable that the child had obtained infection from the mother in early childhood, and that thus the case was really after all one of acquired syphilis. A certain number of infants and young children do, in accidental ways, become infected, and of these I much suspect that the patient we have just seen is one.

Raynaud's Phenomena in an Elderly Woman.

Our patient is a single woman, aged 62. She tells us that in childhood she used to suffer from chilblains on her hands and feet, but not more than other children. She went into service young, and as housemaid, dairy woman, and subsequently cook, has led a very industrious life. She has usually enjoyed good health, and through all the middle periods of her life has been accustomed to put her hands into cold water very often, and

without any inconvenience. Her statement is that early one morning, in April of 1888, after an exposure to cold not at all more than usual, she was seized with severe pains in her hands and arms. These pains recurred afterwards every morning, and were attended first by pallor and then by duskiness. She used to relieve them by drinking warm tea. Gradually, however, the pains would last longer, and the hands would become more livid. Subsequently she became quite unable to do her hair up in the morning, or to feed herself at breakfast, on account of the numbness or pain. At length she was obliged to wrap her hands in cotton wool in order to keep the fingers comfortable, and to prevent their becoming livid. On one occasion small patches of skin on the tips of all the digits of the right hand sloughed. Her condition at present—a moderately warm day and a very warm room—you see. All her fingers are slightly swollen, of a bluish tint which increases in depth towards their tips, and quite cold. Her nails are weak, fibrous, and show transverse ridges. There is not the slightest atrophy or tightness of the skin. Her feet are in a similar condition to her hands. In all other respects she is in good health.

Comments.—We have in this case a typical example of Raynaud's malady, *i.e.*, of exaggerated and quite morbid susceptibility on the part of the extremities to the influence of cold. The slightest exposure induces closure of the arteries, which is rapidly followed by fulness of the veins. The condition, although seldom completely absent, is distinctly paroxysmal, as it ought to be in all real examples of Raynaud's malady. The case is a little exceptional on account of the age and otherwise sound health of the patient. I am very glad that Dr. Sturrock has given us the opportunity of demonstrating it, because it allows me a good opportunity for asking attention to the differences between such cases as this and two other forms of symmetrical gangrene which may be confused with those to which Raynaud's description properly belongs. One of these forms I may almost claim to have first described in connection with these demonstrations. At any rate we have collected for the first time a group. We have had here for demonstration repeatedly a man who lost the terminal inch of all his digits, hands and feet, the tip of his nose, and the borders of his ears. In the museum is a portrait showing his condition during the stage of gangrene. Symmetrical gangrene of the extremities—acro-

sphacelus—it was indeed, with a vengeance; but it was not Raynaud's malady. There had been nothing paroxysmal in the history of the case. The man had had one single severe illness. From this he had recovered with the loss of the parts named, and his circulation ever since has been quite stable. For some time I thought this case unique, but the zeal of one of my audience, Dr. Connor, discovered another which had been published, and quite recently I have myself found a third. The three are all alike, all in men, and in all the recovery of the circulation has apparently been complete. It is never so in true Raynaud's disease. The other group of cases, which we must distinguish, is one in which the skin passes into a hidebound condition, to which the term diffuse morphœa, or sclerosis, or scleroderma, may be given. A number of these cases have been described as examples of Raynaud's malady; indeed, one or more are related in Raynaud's original treatise. You will observe, however, that in the patient before us there is not the slightest approach to scleroderma. The phenomena which we witness are simply those which Raynaud designated by the term asphyxia. It is a paroxysmal narrowing of arteries with dilatation of veins, their consequences and nothing more.

The cases of diffuse scleroderma must be put in a different category. They are attended with liability to paroxysms, and with proneness to gangrene; but these are complications of the hide-bound condition. I do not believe that the liability to paroxysmal asphyxia ever leads to scleroderma, however long it may last. The case before us is a good instance of this, for the patient's hands have been a good deal disabled for eight years past, yet there is not the slightest approach to the hidebound state.

I will just give one hint as to the treatment of these conditions, more especially when they occur, as in the present instance, in patients who are past middle life. The remedy which, in my experience, is by very far the most effectual, is the habitual use of very small doses of opium. I usually prescribe from three to five minims of Battley's liquor three times a day, and the effect is that, without inconveniencing the patient in any respect, it greatly favours the circulation in the extremities and adds much to the comfort of life. If the patient be wealthy, some wines, more especially Madeira, appear to have much the same effect. Opium in minute doses is, however, more easily obtained, quite as effectual, and probably as little liable to abuse.

Progressive Muscular Atrophy.—Comments on a case.

Dr. Hughlings Jackson has been good enough to send us from the Queen's Square Hospital an excellent example of this remarkable malady. The patient is a man, aged 48, who has served in the army. Were it not for his paralysis he is in excellent health. He is willing to own to syphilis twenty-two years ago, but as he says he had suppurating buboes and no eruption, that he took no mercury, and that he has had no reminders, we cannot feel certain that he had anything more than local sores. Although progressive muscular atrophy has been repeatedly observed in connection with a syphilitic history, yet the exceptions have been so numerous and definite that we cannot hold that the disease itself constitutes any strong presumptive evidence of taint. Our patient tells us that his symptoms began about two years ago by what he calls dropping of the left wrist. Next his right wrist was similarly affected, and a year later the muscles of his upper arm and shoulder began to fail. At present the conditions are almost symmetrical, but are a little more marked in all the muscles of the left limb than the right. I will not attempt to go through an enumeration of all the muscles; but we may note that the conditions do not suggest the affection of special nerve trunks. Thus in both hands he has lost the power of separating the digits, but there is not the hollow between the thumb and forefinger, or the atrophy of those thumb muscles supplied by the ulnar nerve, which would denote paralysis of the latter. In both arms the brachialis anticus appears to be quite atrophied, whilst the biceps is of good power. This gives to the arm a most singular appearance when it is bent at the elbow, for the biceps muscle and tendon have nothing beneath them, and stand out at apparently an inch distance from the bone. The deltoid on both sides, and the serratus magnus on both are, as far as we can tell, entirely atrophied. His right trapezius is weaker than the left. The sterno-cleido-mastoids are not as yet materially affected. Of other nervous symptoms, with the exception of the eyes, there are none. There is, of course, no defect of sensation anywhere, and no secondary lesions of nutrition. It is muscular paralysis, and

nothing else. The man can, by steadying one hand on the other, just manage to write his name. His eye symptoms consist of a condition of extreme myosis, but he has no loss of accommodation, and can read easily. I am not certain that his pupils should be described as the Argyll-Robertson, for there is, I think, a little dilatation when the eyes are shaded, and the differences between accommodation and the reverse are very slight.

A CASE OF

RELAPSING CATARRHAL INFLAMMATION OF THE VERMIFORM APPENDIX.

TREATED BY OPERATION.

By ANTHONY BOWLBY, F.R.C.S.,

Assistant-Surgeon to St. Bartholomew's Hospital.

T. M., aged 19, was admitted into St. Bartholomew's Hospital on November 14th, 1893. He was sent from Brighton by Dr. Greenyer, who kindly supplied the previous history, which was as follows:—The lad had always enjoyed good health until June of the present year, when, after heavy work, he was laid up with gradually increasing pain in the right iliac fossa. An examination revealed a painful and tender swelling in this region, and there was slight pyrexia. The bowels were confined but there was no sickness. The pain lasted for eight or nine days, and then gradually subsided under the use of fomentations. The patient was soon able to return to work, and considered that he had quite regained his usual health. On August 17th, however, he was suddenly seized with a return of his previous pain whilst on the beach, and was laid up for ten days. At the end of that time he was able to be up and about, but could not return to work as any active exertion brought on a dull aching in the groin. On September 26th, in the evening, the pain returned with great severity, becoming very acute and spreading over the whole abdomen. The next day he had symptoms of acute peritonitis, with sickness, retching, and distension. There was some pyrexia and slight delirium. The bowels were confined, and there was pain on micturition. Under careful treatment this attack gradually subsided, although the patient afterwards had a dull aching sensation in the groin, and was unable to take any exercise on account of increase of pain. The bowels remained confined, and were only opened by enemata. It was after this attack that Dr. Greenyer considered that an operation was advisable and sent the patient to London.

The day after his admission (November 15th) I examined him. When lying in bed he had no severe pain. The abdomen moved well on respiration, and no swelling could be seen. On palpation there was slight tenderness half way between the anterior superior spine and the umbilicus, but no swelling of any kind could be detected, and the muscles offered no resistance to deep pressure. *Per rectum* nothing abnormal was felt, but there was some slight, though definite tenderness on the right side of the rectum. The viscera appeared otherwise normal.

On the 18th, Mr. Thomas Smith and Mr. Marsh saw the patient in consultation with me, and agreed that it was advisable to recommend an exploratory operation. I therefore arranged to perform this on the 24th, keeping the patient in bed meantime, and emptying the intestines thoroughly on the 22nd. The operation was performed with the kind assistance of Mr. Marsh, the abdomen being opened by an incision in the right linea semilunaris. On introducing the finger into the abdomen, a few slight adhesions were felt at the brim of the pelvis, and then on passing the finger higher up, an elongated swelling was felt an inch above a line drawn from the umbilicus to the iliac spine, and behind the cæcum. The incision was now enlarged upwards, and the cæcum drawn inwards, when a greatly enlarged vermiform appendix was exposed. It was about the thickness and length of the little finger, and lay with its free extremity upwards. It was fixed by adhesions to the cæcum and the iliac fossa, but the cellular tissue in this region was quite normal. The end of the appendix was freed without trouble, its mesenteric attachment was clamped, divided with scissors, and ligatured, and the appendix was then removed. The removal was accomplished by dividing the peritoneum and retracting it (as is done with the skin in a circular amputation), and then tying the denuded appendix, the peritoneum being subsequently stitched over the stump. The wound was closed with buried

silk sutures, and dressed with iodoform and alembroth gauze.

The patient bore the operation without suffering from shock or pyrexia, and ten days later, on removing the dressings, the wound was found soundly healed. After rest in bed for three weeks he returned home.

An examination of the appendix after removal showed that the peritoneal coat was greatly distended. It measured three inches in length, and was very firm and slightly elastic. On laying it open, its lumen was found to be very much narrowed by a general swelling of its mucous membrane. It contained a very little viscid mucus, but no fæcal matter, and no concretions.

This case was evidently one of simple catarrhal inflammation of the vermiform appendix. The cause of such a condition is very doubtful, but it is evident that when once swollen by inflammation an organ attached by so narrow a base and so slender a mesentery must have some difficulty in emptying itself of the exuded inflammatory products. The swelling thus becomes chronic, and the distended organ, now in an unnatural condition, more easily becomes again inflamed. It is probable that as a result of several such attacks the vessels in the mesentery of the appendix become compressed by fibrous tissue and interfered with by adhesions, so that a later acute attack may result in arrest of the venous blood stream, and thus cause a moist gangrene. To operate before so serious a result has taken place is evidently the safer plan, and the conditions found fully justified Dr. Greenyer's advice to his patient to submit to operation forthwith.

With regard to the incision employed, I would remark that in cases where there is neither thickening, swelling, or tenderness in the iliac fossa, it may fairly be inferred that the trouble is mainly intraperitoneal, and that when this is the case an incision in the linea semilunaris is better than one nearer to the iliac spine. By the former incision the appendix can almost certainly be reached in whatever situation it may be found; whilst it is difficult to reach it by the latter incision if it be at all abnormally placed, unless by a very extensive division of the muscles of the abdominal wall. In any case, an incision in the linea semilunaris is not so likely to be followed by hernia as one placed nearer to the iliac spine; and for this reason alone is, I think, to be preferred to that which is more commonly employed.

A CLINICAL LECTURE

ON

THE REMOVAL OF NÆVI BY ELECTROLYSIS.

By JAMES CAGNEY, M.A., M.D., M.R.C.P.,

In charge of Electro-therapeutics, St. Mary's Hospital;
Physician to the Hospital for Epilepsy and Paralysis;
Assistant-Physician to the North-West London Hospital.

GENTLEMEN,—You have possibly been struck by the variety—I might have said the incongruity in their nature—of the cases which come to be treated in this department. We naturally reckon upon having to do with a large proportion of the nervous diseases properly so called, and by an extension of the term we are prepared to find a medical element calling for special treatment in a number of patients who are sent to us by the surgeons when the resources of their science are exhausted. I need but refer to a patient whom you have seen here to-day—a man, a carter, who was thrown heavily on his shoulder, dislocating it. The displacement of the joint was rectified, and in due course when the swelling and any obvious cause for pain had subsided, it was found that the man was unable to raise his arm level with the shoulder. This might be due to many causes, into which I need not enter. I will single from amongst them two of very different significance, namely, malingering and a secondary paralysis of the deltoid muscle. Both incidents are among the common sequelæ of injuries to the shoulder, and they are hard to discriminate. I will go farther, and say that it is impossible to distinguish between them otherwise than by the diagnostic aid of an electric battery.

Such a case, then, is properly grist for our mill, and falls naturally within the province of the physician, more especially since, as I showed in this instance, he will generally find that the patient calls for his aid in more ways than one, and derives benefit therefrom. But in the series of cases which I have according to custom reserved to the last, and to which we shall address ourselves, it is different; no good reason can be assigned for their coming here to us who claim to be physicians and not surgeons; they are purely surgical cases.

You will observe that they are all nævi, and probably you will find on inspection that they are what I may call healthy nævi. Here and there

is found one which is scarred on the surface, showing that it has received desultory attention from the practitioner; occasionally, the nævus is sloughing and covered with granulations, possibly promoting its own cure in its own way; more probably while breaking down at the centre, it is creeping serpiginously at the edges, making its way along the folds of the fat and dimpled skin, and threatening a lamentable destruction of tissue. Such an one has come to us none too soon, but it is the exception. For the most part these nævi are simple vascular tumours, exhibiting no pathological element but that which belongs to their own nature. Therefore I call them healthy, and they grow mostly upon healthy children — the mothers are careful about this. The habit of maternity, as you are well aware, generates an ungrateful suspicion of the workings and purpose of nature. Above all, the maternal mind is apt to brood on cancer and to believe that it lurks behind every passing ache, and puffs out the smallest swelling. The mother's anxiety extends to her children. The sight of a nævus on the newly born infant appals her, and at the earliest opportunity she takes advice upon it. The children have not had time to grow ill. They are mostly from one to twelve months old, when they come here to have the growth removed. But why here? is a question you naturally ask yourselves. An electro-therapeutical department must be thought from its very name to concern itself with therapeutics, legitimately too with the diagnosis of neuro-muscular ailments, but not at all with surgical interference; and yet I believe it is the rule in metropolitan hospitals to relegate the operation for nævi to the physician who has charge of electro-therapeutics. The explanation which I would offer is a little humiliating, but in the cause of science and for the incidental advantages it implies it is to be borne. I wish to-day to persuade you, each of you in his own practice, to electrolyse your nævi for yourselves; I want to show you that a battery is not the perverse thing that those who know nothing of its construction believe it to be; that with a very little knowledge it is easily manageable, and that if it can be shown that it is eminently useful, even indispensable, to the surgeon, it is discreditable to the surgeon to be without this knowledge. For the present I must point out to you that it is because this knowledge is far from general that the electro-therapeutical work in the hospitals is made to include a certain share of surgery. It is in vain that such men as my predecessor here, Dr. de Watteville, have won their reputation in the treatment of disease; that Prof. Erb is at once the leading authority upon the use of electricity in medicine, and at the same time is one of the most illustrious physicians of the age; in vain that the enlightened labours of many other able and enthusiastic men in every country has brought and is still bringing electro-therapeutics to the position of a science at least as exact as that of any other branch of medicine. In spite of that, we, who who profess it, are known to most of our contemporaries as "electricians." We are electricians because we understand the use of a battery, and others don't. I dare say if one of our great surgeons happened to know how to sharpen his own knives, and in this had the advantage of his *confrères,* he would have a preponderant reputation as a "cutler."

But the confusion of ideas is to be regretted. However, like the gift of the god to the blind bard in the Odyssey, it has in it both of good and evil. If we are electricians, the masseuse who is on friendly terms with an electrical instrument maker is, for practical purposes, an electrician also, and in that capacity she is allowed to work her undetermined will upon nervous patients, with a result that is often to be deplored; and I have heard of highly-educated physicians who similarly employ the leisure hours of the instrument maker himself, and are so developing a new branch of medical practitioners. This is a pity; it is not perhaps creditable to the profession; and it is open to question whether it is just to the public. These are the disadvantages of the case. Among its advantages is prominent the fact that as electricians —that is, as men who, whatever else they know, know also how to keep their batteries from being short-circuited—we are provided with organic material to electrolyse, and notably with nævi. With this digression, which I offer as an apology for what I always feel to be an intrusion upon the sphere of others, I shall betake myself, *in medias res,* and speak of nævi, and of the way in which we remove them here.

Characters and distribution of nævi. I shall confine myself to a very few remarks on this head. Their purpose will be to enable you to discriminate the cases in which electrolysis is to be recommended and to discern the steps which should be taken in applying it. Nævi are a very

common disfigurement. Depaul stated that one-third of the infants born at the clinic of the Paris Faculté of Medicine are blemished by them. They occur elsewhere than on the surface of the body, but for the most part form in the corium of the skin or just beneath it. In either case they give rise to disfigurement, and that is their chief significance, but they are not without a degree of danger. In many cases they disappear of their own accord, but their tendency to do so is, I think, over-stated. On the other hand, they may give rise to serious bleeding, or they may break down and slough with the absorption of poisonous products. I have known instances in which the process of sloughing at central portions of the tumour was accompanied with an extension of the growth peripherally at a rate so rapid as made it extremely difficult to check it. T. Müller has recorded cases of malignant cavernous angiomata with a tendency to increase and produce secondary growths.

Nævi are of two kinds according to Cornil and Ranvier's classification. (a) Simple angiomata (congenital nævi). These are most common about the face and neck, and they involve the skin or subcutaneous tissue. They may be imbedded in the skin or project from it. They are scarlet, purple or blue in colour, and they consist essentially of a new formation of quite normal vessels for the most part capillaries of exceedingly delicate walls, and marked here and there with ampullar or cirsoid dilatations.

(b) Cavernous angiomata (venous nævi; erectile tumours). These, too, occur for the most part in or beneath the skin; they are found also in the orbit and on the surface of viscera (liver, kidneys, spleen, and intestine). The tumour is essentially a cavernous system, resembling normal erectile tissue, diffuse or circumscribed, or even encapsuled. There is a relatively abundant fibrous stroma, and a considerable quantity of fat may be entangled in its meshes. Cavernous nævi are generally bluish in colour. They sometimes pulsate, and may be temporarily reduced by pressure.

According to their situation, superficial nævi may be distinguished as cutaneous, subcutaneous, and mixed. Cutaneous nævi again may be a mere staining, the " port-wine mark," or a distinct mass of larger vessels. In the first the vessels are exceedingly small and uniformly distributed. You will do no good with electrolysis here. So many punctures would be needed, and these must be

so close together that the operation would be infinitely tedious, and it would be effective only by the wholesale destruction of skin. The galvano-cautery serves better.

The so-called "stellate nævus," on the other hand, which causes a somewhat similar but more limited staining, yields readily and surely to a very trifling manipulation. The other nævi which form definite swellings, whether in, beneath or upon the skin are, I believe, in all cases suitable objects for electrolysis.

The process of electrolysis. You are all familiar with the phenomena of electrolysing water. You know that when two platinum points (electrodes) carrying the poles of a constant current are placed apart from each other in a vessel of water, the water is decomposed into its constituent elements, oxygen migrating to and being given off at the positive pole, and hydrogen at the negative; and if the current be allowed to continue, the process goes on until all the water is decomposed. The water is called the electrolyte. All fluids that can conduct an electric current are electrolytes—that is, they can be decomposed by the current, and they conduct probably in virtue of this property. If to the water be added an acid or a salt which it can dissolve it has more of the character of an electrolyte: it conducts better, decomposition is more rapid, and its products include the elements of which the acid or the salt consists—the oxygen-like element of the compound passing to the positive pole and the hydrogen-like element to the negative. In this respect all the elements of an electrolyte or conducting solution have a relation to one another comparable to that between oxygen and hydrogen in distilled water. If, instead of platinum points, steel or another metal which readily combines with oxygen, had been used, a new compound (of iron and oxygen) would be formed at the positive pole during the decomposition of water; and so if the electrolyte had been a dilute solution of hydrochloric acid a steel needle at the positive pole would be eaten away gradually with the formation around it of oxychloride of iron. It is simply to prevent this new formation that platinum is used. Gold serves the same purpose. The secondary compound into which the metal may enter is no part of electrolysis; but it may be utilised in the electrolysis of nævi, as we shall see. I do not propose to go any further into the physics of electrolysis. It has its theories, of course, and it is but one phase of the fundamental laws which

underlie the whole science of electricity, but those things do not concern us here. I will only add what you have already anticipated, that the blood, serum, and organic fluids of the body generally are electrolytes, and so can be decomposed by the passage through them of a constant—but not of any other—current. Again, the more fluid, the more vascular, any tissue, the more readily is it decomposed. The solid tissues are not decomposed at all, in the sense that the electrolyte is; but you know that the abstraction of fluid from a soft tissue implies the destruction of that tissue, and in this way even the solid parts are destroyed by electrolysis, and it is only in this way that they are destroyed. I believe the impression is common enough that electrolysis acts like an escharotic or a cautery. If anyone happens to hold this view it is most important that he should get rid of it.

Requisites for electrolysis. 1. The most important instrument is the battery. It must, of course, be a constant current battery, and medical men will use one of a form which is most generally useful —that is, which can be applied to the greatest number of their purposes. It is on that ground that we use Leclanché cells here. A battery is a collection of cells, but the cells may be coupled up in different ways to suit the work that the battery has to do. To heat a wire for the galvano-cautery, for instance, certain cells are preferable to others, and they must be connected in a particular way. For ordinary medical work and also for electrolysis any cells will do, and they are connected in what is called "series." Ten to forty Leclanché cells in series constitute your battery. A smaller number of Daniell's or Bunsen's will do. Hellesen's dry cells might be employed, or an accumulator of sufficient capacity.

I cannot enter here into the considerations that would guide a choice of cells in special cases. Generally speaking, the advantage of one kind of cell over another is an economical advantage, and to secure it requires an elementary knowledge of electrical physics. The underlying principle has to do chiefly with resistance in the circuit, and it may be stated broadly that a given number of cells will yield the strongest current when the resistance within the battery most nearly equals the external resistance. It will be seen that the latter varies greatly with circumstances, as with the nature of the growth to be electrolysed, the position of the electrodes, and so forth. The cells should if possible be large, as this lessens the internal resistance;

it is most important where both poles (needles) are inserted beneath the skin. Finally, where a surgeon desires to fit up a battery for the purposes of electrolysis alone, bichromate, Hellesen, or Bunsen cells are the more economical.

2. A galvanometer should be included in the circuit. By its means not only can the current strength be accurately determined, but its diminution by polarisation can be watched and remedied, and the danger of short circuiting by accidental contact of the needles is guarded against.

3. The battery should be provided with a cell collector by which any desired number of cells can at any moment be thrown into the circuits, and with an alternator, a mechanical arrangement by which the positive pole can be made negative, and the negative positive without removing the electrodes (needles).

4. The electrodes. These are generally needles, connected with the positive and the negative poles, and in both cases they are usually plunged into the tumour beneath the skin. This is the quickest and most energetic mode of operation. There may be one or more needles attached to either or both poles. Where more needles than one are used, the tumour is attacked simultaneously in different parts of its extent. The needles may be of platinum or gold, or of steel. The advantages of the first two are that they do not readily combine with the products of decomposition, and this may, or may not be an advantage. On the other hand, they are costly—excessively costly as they have to be obtained from instrument makers. Platinum again, does not easily retain a sharp point; this is a decided drawback. Steel has the advantage that it is very cheap. Ordinary sewing needles can be fitted into the needle-holders, and since there is no need to resort to an instrument maker for them, an assortment of various sizes can be had for almost nothing. It is only in the case of that one which carries the positive pole that any disadvantage at all attends the use of steel needles. That needle will corrode and rust, entering into combination with the oxygen, chlorine, and other gases set free at the positive pole. In consequence, it becomes adherent to the tissues, and cannot easily be withdrawn; there is a remedy for this however. It is destroyed in the process, and must be thrown away afterwards; in view of its cheapness this may be disregarded. The deposition of fixed substances upon and around it takes from the current strength; up to a certain point this

may be met by throwing in more cells. The chief objection to the use of a steel needle as the positive electrode is in this, that its decomposition stains the tissues, and by infiltrating them at the point of its insertion with inorganic matter it tends to cause sloughing. The tendency to the sloughing however is in any case very slight, and the stain if it does not slough out disappears in time.

On the other hand, the decomposition of the needle provides a powerful hæmostatic which is often of the utmost service. On the whole, more can be said for it than against it.

Both poles need not be inserted into the tumour, one may be carried by a plate electrode of large size applied to the surface of the body, usually over the sternum. In this way the external resistance is raised from a very slight to a very great one, and a greater number of cells will be needed; the process is also slower and less energetic. The method will be adopted where as in the case of a small stellate nævus a single puncture will do what is required, and to avoid all possibility of a scar, the puncture will be made with the negative pole.

The operation is intensely painful, and will rarely be done without an anæsthetic.

The operation of electrolysis. This is essentially the same, whether one or more needles be used for either or both poles. In the great majority of cases it is our custom here to use two needles, fixed in an insulated handle by a small screw. The child being anæsthetized, both needles are plunged into the substance of the tumour and cells are thrown into the circuit until the galvanometer records a current strength, such as may be deemed desirable. This may be from 20 to 40 milliampères. The stronger the current the more rapid and energetic is its action, and the greater the chance that sloughing will take place afterwards. The desirability of avoiding this contingency will depend largely on the situation of the nævus, but not entirely. It is a matter of observation that large sloughs will separate after electrolysis, and yet a healthy skin will grow in their place and scarcely any scar will remain after the lapse of months or years. On the other hand, when a nævus has been electrolyzed with a weak current, and sloughing has been carefully avoided, the marks of the punctures have remained; and if numerous, their scars may persist long, or always, an unsightly memorial of the operation. These scars, in my experience, are less likely to disappear than are the signs of more wholesale but temporary

skin destruction. Thus, while the surgeon will study by every means to lessen the chances of disfigurement when its situation would be on an exposed part, such as the face, he will not attain his end in all cases by undue caution. In such situations he will be more careful. He will watch the process narrowly, and experience alone can teach him the proper management of his current. Where the nævus is on the trunk or part of an extremity that is usually covered, he will proceed more boldly, employing a strong current, changing the situation of his needles frequently, and finishing more rapidly. In such cases, too, he will often get his best results. I have said that experience is the necessary guide.

While the current passes, bubbles of gas are given off freely from the point where the negative needle penetrates the skin. This needle remains brightly polished, and it moves easily in the puncture. The positive needle, on the other hand, becomes clogged with a blackish rust; for a short space around it the skin grows dark and hard, and if any blood exudes slowly, it is coloured nearly black. The needle itself becomes firmly adherent to the tissues around, and can hardly be moved without tearing them. For this reason it is moved as little as possible. Two or three central spots are chosen, into each of which it is driven deeply and allowed to remain a relatively long time, while the negative needle is constantly withdrawn and replaced in the substance of the tumour. Where the risk of sloughing may be faced, the positive needle may be made to transfix the base of the nævus, and to remain there while the negative is applied successively to points all over the surface. Both needles must in every case be driven well into the vascular tissue. If a large artery can be seen, as it sometimes can, to enter the nævus at a particular spot, the positive needle may be passed through this with good hope of breaking its continuity by clot or destruction. Meanwhile, as the process of electrolysis goes on, the tumour is converted piece by piece into a doughy mass, rendered more or less emphysematous by disengaged gas. There is a state recognisable by the sense of touch which proclaims that enough has been done. To go farther in the direction of hardness renders sloughing imminent, and when this state is reached the operation must end. This is the object which alone is regarded in the more uniform tumours—the simple angiomata. In the case of cavernous nævi, or where

distinct vessels can be made out, there is room for skill and judgment in efforts directed to the destruction of the latter, or to forming clots in particular situations.

When it is decided to withdraw the needles, it will be found that that carrying the negative pole comes out easily. To remove the positive the poles are reversed by means of the alternator while the circuit is still made, the current is allowed to continue for a short time, and the rusted needle, which is now negative, frees itself by the formation of gas around it, and can be easily taken out. Bleeding of course is not uncommon, but it is always under control. It is most apt to occur at the positive needle when the latter is hastily removed. If, however, the needle be put back again, and more slowly withdrawn so as to electrolyse the blood as it flows behind it, the puncture will be plugged. If this is not sufficient, a turn of the alternator makes the needle positive, and salts of iron are instantly formed by its decomposition. In this way a most powerful styptic is instantly available, and applied with unerring accuracy to the injured vessels. Should bleeding continue when it is desirable to cease electrolysis, a pledget of cotton wool soaked in collodion may be applied as the needle is withdrawn.

If a nævus is very large, it may be inexpedient to destroy it all at once. It can then be done in parts separately. The surgeon will endeavour, by appropriate means, to destroy the vascular tissue thoroughly, either mediately by clot and the destruction of vessels, or immediately by electrolysis of the mass—most often in both ways combined; but he will be on his guard against doing too much. He can finish later what he has left undone in the first operation. And in any case it is better to do too little than too much. Time tends to complete the cure.

The after-treatment is simple—no dressing required. A covering to keep the part clean will meet the necessities of the case at first. Nothing more may be needed; but the patient should be seen in a day or two, and if there be any sign of sloughing, the wound should be dressed. At the end of a week it will be covered with healthy granulations, which only require to be washed with boracic or a similar lotion. I have not met with any complications of a septic character. I believe they do not arise after electrolysis of a cutaneous nævus. My assistant, Dr. Nathan,

informs me that a considerable number of the infants upon whom we operate, suffer from diarrhœa during one or more days immediately following this operation. We have not found any cause for this. I am constrained to add one word on the subject of anæsthetics. A distinguished writer on the uses of electrolysis has said that it is dangerous to give an anæsthetic when tumours about the head (as in the scalp) are to be electrolysed. I have had the opportunity in many hundreds of instances to see the signs of this danger if it existed, and I never have seen it. Theoretically I cannot conceive any grounds for it. The administration of an anæsthetic is nearly always indispensable, and I do not hesitate to say that it is free from any special element of danger.

The advantages of electrolysis. I have already expressed my belief that it is only the contemporary ignorance of physics—the want of knowledge, which implies the want of skill in the use of apparatus, that causes electrolysis to be other than the routine treatment of nævi. It is true that I have little knowledge of the surgical alternatives, but it is possible to reason *à priori* in the matter, and my judgment, it seems, must be that of others, if I can base an inference on the great frequency with which nævi are sent to this department to be removed. Electrolysis seems to me to have the following advantages: It is bloodless, its efficacy is certain, it is quickly over, it is entirely free from dangers to which surgical procedure is always exposed, it leaves or need leave no disfigurement, and it aims at and should succeed in destroying nothing but the growth which we desire to remove. Indeed, it appears to me to involve more of the fitness of things than any surgical procedure whatever, for while the latter aims at removal in bulk, electrolysis has the more ideal end of dissipating superfluous matter, first into its elements, and then into nothing. There is in this a certain philosophical perfection which may even seem to you to be transcendental and beside the mark. But the perfection is the perfection of means also.

The "Lancet" recently reported a lecture by Mr. Pridgin Teale, a very great authority, in which he dwells upon the incidental advantages of cicatrix. He points out how a scar having a tendency to contract as time progresses, may be so utilised as to obliterate the channels of blood supply, and so to starve a growth dependent on

them; and Mr. Teale justly claims for such an expedient a very high place in the surgeon's esteem. He says, "the principle of utilising cicatrix has been my constant guide in the treatment of nævus, more especially of the small superficial ones about the face." But the cicatrix that can be caused by cutting with the knife is as nothing in comparison with that which may be made to pervade the whole structure of an angioma by the conversion of its soft parts into fibrous tissue under the electrolysing influence of a constant current. I also have long had it in my mind that it is this agency of cicatrization which must be utilised to strangle the nævus; but I try and secure a cicatrix other than that which marks the track of the knife—a finer and more subtle scar which can be strengthened and accentuated at one part, attenuated at another, but made to extend throughout the growth as a web-like structure endowed with the power of contractility and destined with the lapse of time to close in upon and destroy all trace of excessive vascularity. The formation of this pervading scar must, I submit, be the result of judicious electrolysis, and hence it happens that the nævus which has been electrolyzed tends progressively to disappear. It is a thing commonly to be seen that one of these tumours which seemed some weeks after the operation to be little less in bulk than formerly, and still in patches decidedly vascular, has gone on from a month and even, it is said, for many years, growing always and "more beautifully" less, to disappear entirely before the infant has attained to puberty. It was for this reason, it was on account of the sure co-operation of time, that I gave you the caution to do little rather than too much; and I would add to this caution the further advice, not if you can help it, to repeat your operation for many months after its completion.

It remains to ascertain whether there are any cases or any forms of these angiomata which may properly be returned to the surgeon as being unsuitable for electrolysis. Now that is a question which I am embarrassed to answer. I have seen nævi certainly which I would rather not interfere with, and a few which I have declined to touch. These are usually complicated with some unhealthy condition which is unfavourable. I hasten to mention that sloughing is not such a condition. On the contrary, given a nævus which sloughs and threatens to extend widely into the healthy skin,

electrolysis is for such an one imperative and possibly the only remedy. I say this with deference as one who has little knowledge of the alternatives; and my ignorance upon this head is the chief difficulty in the way of discriminating the cases which can least be recommended for electrolysis. There are methods in use which under any circumstances strike one as barbarous and unscientific. The application of acid and vaccination upon a nævus seem to be such; and if to decline to electrolyze were to give the preference to one of these, there could never be any hesitation. Again, the great size of a tumour does not make it more difficult to destroy in this way, whereas I suppose it renders other expedients more formidable. Nor, again, is the great size of the entering vessels a bar. Electrolysis has given some of its best results in cirsoid aneurysm.

There is, however, a form of cavernous angioma, in which the question of excision may profitably be entertained. This form is of not very common occurrence, and can generally be recognised. I have seen it most often about the forehead or scalp, and particularly in the neighbourhood of the orbit. It is usually fed by one or more large vessels. The cavernous spaces, I think, are large also. The blood can be expressed from such a nævus as this by pressure of the finger, and under those circumstances there is felt beneath the skin a considerable bulk of tissue which gives a semi-elastic boggy impression to the touch. This is the fat-laden stroma of the nævus. Not much can be done with it. Its fluid contents can of course be electrolysed, and clots no doubt are largely formed in the process; but one is prepared to find as a result, that the size of the tumour is but little lessened, and the subsequent process of scar contraction has slight effect. Absorption to some extent doubtless takes place in time—the child grows and the tumour does not—it rather shrinks. This much can be done by electrolysis, but if a scar can be borne, the knife will do more. If the growth be doubtfully one of this sort, or if it be thought well to try electrolysis first, then the punctures should be made in the line of the future incision, so that the ultimate disfigurement shall not be greater than if the knife had been used at the outset.

Ulcer of Stomach.—Bismuth sub-nitrate ℥iiss, fasting, in morning, followed by water f ℥iss.

(Berliner Klinik.)

THERAPEUTICAL NOTES AND FORMULÆ.

Resorbin.—Dr. R. Lederman of Berlin draws particular attention to this substance as a specially useful basis for ointments and salves. It distinguishes itself particularly by the extraordinary power it possesses of penetrating the skin, so that it rapidly disappears with very slight rubbing-in, leaving only a little greasiness of the skin. On account of the water it contains it acts as a cooling agent, diminishing itching and inflammation. It is therefore peculiarly adapted for use in pruritus nervosus, especially the senile form; also for prurigo, as basis for naphthol or sulphur ointment. On the other hand, it is not applicable in deep skin affections with free secretion, e.g., eczema madidans and suppurating wounds. It is very useful to remove the scales and crusts of eczema impetiginosum, of psoriasis and seborrhœa sicca, more so if used in combination with salicin or sulphur. It is, moreover, useful in the treatment of ichthyosis and artificial dermatitis. In combination with boracic acid, salicylic acid, sulphur, oxide of zinc, white precipitate or tar, it is useful in subacute and chronic eczema; here it is valuable as a preventer of adhesions, owing to the greasy surface that it leaves. For inunction in syphilis it has advantages, carrying the mercury in very rapidly. Pruritus vulvæ has been found by Dr. Hahn rapidly to yield to resorbin.

(*Dermat. Monatschr.*)

Diphtheria. — Treatment at Hahn's clinic, Berlin: Application of ice collar, hourly gargling or spraying with 4 per cent. solution of chlorate of potassium, or 1 to 4,000 solution potassium permanganate in cases where fœtor is present. In nasal diphtheria, syringing of nasal cavities with 2 per cent. boracic acid or 1 to 4,000 permanganate solution, plugging nostrils if epistaxis occur. Steam in cases of laryngeal obstruction. If urgent dyspnœa, low tracheotomy, removing cannula on fifth or sixth day.—(*Deut. Med. Woch.*)

Djamboe.—Of most value in infantile diarrhœa. Used in some hundreds of such cases with success, also in diarrhœa of phthisis. Dose gr.7½; larger doses without bad effects. Infusion preferable for children, fluid extract for adults.

(*Munch. Med. Woch.*)

Alopecia Areata in Children.—Cut off hair with scissors as closely as possible; apply following ointment every evening:

℞ Vaselin	⎫	each
Fresh lard	⎭	ʒʒ¾
Precipitated sulphur		gr.xlvj
Salic. acid		gr.xvss

Next morning wash head with salicylic acid soap, brush with soft brush dipped in

Alcohol		f ʒʒ¼
Tinc. rosemary ...		f ʒʒ¼
Sublimate		gr.ivss

Once a week paint affected areas with

Ess. wintergreen ...		ʒiiss
Ether...		f ʒiiss

(*Jour. de Méd. et de Chir. Prat.*)

Sickness of Pregnancy.—The following three formulæ have all proved themselves useful in the hands of various contributors:

℞ Cocain	.03 gram	
Antipyrin	1	„
Aq. Destill. ...	120	„

Sig.: A teaspoonful every half hour.

℞ Tr. Iodi.	7.5 gram	
Chloroform ...	60	„

Sig.: ℳv in seltzer water morning and evening.

℞ Cocain Mur. ...	1	gram
Extract. Belladonnæ	.25	„
Vaseline	15	„

Sig.: To be smeared on the os uteri twice a day.

(*Deut. Med. Woch.*)

Tobacco Habit.—

℞ Gold and sod. chlor. ...	gr.$\frac{1}{24}$
Strych. nit.	gr.$\frac{1}{60}$
Nitroglyc.	gr.$\frac{1}{300}$
Atrop. sulph.	gr.$\frac{1}{300}$
Tinct. digital.	ℳiij
Capsicum	gr.$\frac{1}{4}$
Salicin	gr.j
Cinchon. sulph. ...	gr.j

For one pill.—(*Therap. Gas.*)

A CLINICAL LECTURE

ON

OPTIC NEURITIS.

Delivered at the National Hospital for the Paralysed and
Epileptic, Oct. 24, 1894,

By W. R. GOWERS, M.D., F.R.S.

Physician to the Hospital and Consulting Physician to
University College Hospital.

GENTLEMEN,—My task to-day is to endeavour to help you to help yourselves. There are some things that may be taught by another, but there are many things which the learner can only teach himself, and it is with one of these that I want to occupy your attention to-day. I will do so for less than the usual space of time in order that you may have time to learn by observation that which you can only learn in this manner. And I do so because I happen to have the opportunity of affording you the means of observing a case which is almost unique in its characteristics and in the instructive points that it presents. There is one symptom only to which you need attend. The other symptoms are so uncertain in their significance that it is doubtful whether it is worth while for me even to bring them before you for their negative significance.

The symptom which is presented is optic neuritis. The unusual features which it presents are that it is neuritis in one eye only, and that in this eye the neuritis is partial. Any of you can come at another time and examine the patient carefully at your leisure.

You probably know that it is rare for optic neuritis to be one-sided. It is one of the symptoms which usually occur on both sides at the same time, because, as a rule, its cause is either an influence from the brain, which acts equally upon the two sides, or an influence from the blood, which likewise has an influence so general that its effects are bilateral. Whenever you can compare one side that is normal with the other that is diseased, whatever kind of disease it may be, you are always at much greater advantage in your observation than when there is perfect

bilateral symmetry in the morbid change. Many slight morbid changes are difficult to recognize in their absolute degree, and yet not difficult to recognize if there is the opportunity for comparing an absolutely normal state on one side with the slight abnormal condition on the other. .

The case is instructive not only on account of these features, but because the neuritis has developed in a pre-existing condition that is itself abnormal—a condition which depends upon an extreme degree of myopia. This fact adds to its practical utility to advanced students, because a considerable degree of myopia increases the difficulty of observation and the risk of error; and thus the careful study of the case will not only give you definite information, but will also increase your ability to overcome a difficulty which is not uncommonly met with in ophthalmoscopic work, observation under the condition of considerable myopia. As you probably know, this difficulty only obtains in the direct method of examination, in which there must be accurate compensation by a lens behind the ophthalmoscope. I fear I seem to be making the advantages of the case depend on its difficulties, but, in point of fact, every great difficulty that is overcome, lessens a host of minor difficulties. I may further press on you the necessity for careful estimation of the conditions under which you are observing, inasmuch as the myopia here is almost twice as great on one side as on the other. If you try to compensate on one side in the same degree in which you compensate on the other, you will be unsuccessful. I therefore impress upon you the necessity in the first place of always observing and ascertaining what are the conditions under which you are making your observation.

This necessity is very important in all diseases. Students, and I fear sometimes those who are no longer nominal students, listen to the apex of the heart where the apex of the heart ought to be, instead of at first ascertaining where it is. I have frequently known a student fail to find a murmur at the apex of the heart, because he was listening where the apex ought to have been, not where it was. The patient who is before you has a cardiac condition worthy of observation, although

it will not involve that difficulty. There is a loud murmur at the apex, but it extends so widely that you will not miss it, even though you miss the apex and have not done what you always should do—feel where the apex is before you listen. It is a systolic murmur, and there is also a systolic murmur at the base, which is certainly a separate murmur. Never omit carefully to observe collateral symptoms. You will examine this case on account of the condition of the eyes, but it can teach you a good lesson in auscultation of the heart; and if you have another opportunity of examining the patient, you should make a careful examination of the heart. When there is a systolic murmur at the apex and a systolic murmur at the base, the one due to aortic stenosis and the other to mitral regurgitation, it is generally not difficult to ascertain the fact that there are two murmurs if you are sufficiently careful to observe the gradation of the sound, or rather, the sequence of reversed gradation, in passing from the apex to the base. The pure mitral murmur becomes fainter; if it does not, and is even louder at the base and over the aortic orifice than at the apex, you may feel sure that there is aortic constriction, and especially if, as in this case, you can observe a change in the quality of the murmur. I mention this because the co-existence of mitral regurgitation and aortic stenosis, without aortic regurgitation, is not very frequent, and whenever it is met with, it should be most carefully observed, because it is not always, for the reasons I have mentioned, easy to detect.
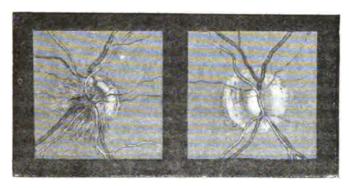
I said that the difficulty involved in myopia is chiefly in the direct method of examination, but a fallacy is caused by myopia in the indirect examination. In this method, the optic disc looks small in myopia, and large in hypermetropia. Just as you should always, before listening to the apex of the heart find out where the apex is, so, before looking into the eye, you should ascertain roughly what is the state of refraction. That is easy to do. First throw the light into the eye by the mirror from a distance, from the distance you would adopt in the indirect method of examination. Move your head a little, and you will quickly catch sight of a vessel. If you see that vessel distinctly, the refraction is not normal; there is either myopia or hypermetropia. Move your head again a little to one side or the other; if the vessel seems to move in the same direction, there is hypermetropia; if it moves in the opposite direction, there is myopia. You then know what to do

when you proceed to the direct method of examination, and you know how to estimate the size of the optic disc as it appears to the indirect method of examination.

This case illustrates the fact in a curious way. I show you large, somewhat rough drawings of the discs, and I have here also a more careful drawing which you can compare with what you see when you examine the patient (figure). In the left eye (on the right, in the figure) the optic disc, of oval outline, appears larger than you would expect. It looks larger than the normal disc, although the eye is myopic, and in myopia objects should look small. It is not the optic disc you see. Note that there are two oval outlines; the optic disc is the inner area, and the outer boundary is simply the limit of what is called a "posterior staphyloma"; that is, an area of atrophied choroid, so common in myopic eyes in consequence of the greater length of the eyeball. The tendency to increase in length leads to a condition in which the opening in the choroid does not reach the opening in the sclerotic, and is increased by a process of atrophy of the choroid.

This case has still further the utility that the arrangement of the vessels is particularly puzzling, and if, with the help of the drawing, you discern them perfectly, you will have enabled yourselves to overcome many other difficulties which you will meet with afterwards in other cases of peculiar vascular arrangement. The peculiarity here is that the central artery is so situated as to entirely conceal the origin of the veins. In the left, the normal disc, you see in the middle the white area of the physiological cup, and the vascular portion of the disc extends up to the place at which the vessels emerge. If you are familiar with normal eyes you will know that that is very frequently the case, especially when the physiological cup is large. The dark portion on the other side of the cup is greyish in tint and not vascular. I have purposely had these diagrams done in black and white and not in colours, for the same reason that I had most of the drawings in my "Medical Ophthalmoscopy" done in monochrome, because it is of considerable more importance to attend to the changes of form than to the change in colour. Nine-tenths of that which is important in the disc depends on change in form—on that which can be shown in black and white, and not in change in colour —and nine-tenths of the errors that are made in

interpreting the appearances are owing to undue weight being given to changes in colour. I should hardly like to say how many cases of normal discs I have had shown to me, as the seat of optic neuritis, simply because the discs were highly coloured. The vascularity of the disc varies as much as does that of the cheek. The test of a morbid condition is not the colour, it is the change in form,—it is especially obscuration of the edge.

In this optic disc, the arteries, as I say, happen to be so placed that they entirely obscure the portion of the veins where the branches unite to pass into the nerve, and it is only on very careful observation that you can discern which are arteries

is a condition of choroidal atrophy, revealing the sclerotic,—a " posterior staphyloma," similar to that which is seen in the other eye. The disc itself begins at the inner oval. As the figure shows, the edge of the disc is distinct on one side, but is entirely lost upon the other. There, you can see no margin to the disc, and you can see no zone in which the white sclerotic is visible in consequence of atrophy of the choroid. It is all covered by a faintly striated, red tissue. On the inner part of this there are several small but distinct hæmorrhages. The amount of swelling is slight. Swelling can be estimated accurately by ascertaining what strength of convex lens behind the ophthalmoscope is required to make indistinct

Partial optic neuritis in the right eye only. Both eyes are myopic. In the right (the patient's left) there is a posterior staphyloma extending all round the disc. The cup is steep on the nasal side (to the right in the drawing), which is of the inverted image, so that in this disc the side further from the other eye is really the nasal, and the darker tint on the sloping side of the cup (apparently towards the other eye, but the temporal) is due to pigment. The emerging mass of nerve fibres extends up to the vessels on the other side.

In the left eye a similar condition is seen in rather less than half the disc; that apparently towards the other eye and therefore really away from it, i.e., the temporal side. The side further from the other eye, that is, the real nasal side, is obscured by inflammatory swelling, which presents also some small hæmorrhages. The clear side, really the temporal, is of course the side turned towards the yellow spot, in which the nerve fibres are few or absent. The figure shows the tendency of neuritis to develop first where the nerve fibres pass off and the interstitial tissue is abundant.

and which are veins. The veins seem to arise from the arteries. It is always necessary to look carefully, to be quite sure, especially in the case of smaller vessels, which is an artery and which is a vein. It is the colour and the character of the central reflection that distinguish them.

Turning to the other eye, we see a condition which is also most instructive. Here also the venous branch is obscured at the beginning by an artery. The upper vein is the most conspicuous object that attracts attention. On looking at the disc, you will be struck by the fact that at one side there is a pale, or almost white zone, pretty sharply margined on the outside, and limited inside by a redder zone. That outer white zone

the surface of the swelling compared with the adjacent retina, or, in the case of myopia, what difference in the strength of concave lens is required behind for distinct vision of each. That is, however, a difficulty in myopic eyes, and you can generally, without much difficulty, estimate the amount of swelling in two other ways. First, whenever there is much swelling, the central reflection of a vein is lost where it descends the side of the swelling. You know the cause of the central reflection from the middle of a vein. That which we call a retinal vein is not the vessel, but the column of blood within it; and from the centre of the convex column of blood the light is reflected straight back

to the eye, whereas from the lateral parts the light is reflected at an angle. The consequence is that all the light is reflected from the central part of the convexity, and this prevents the colour of the blood being perceived as at the sides.

But supposing a vein slopes for a short distance, so that its course is in a plane at an angle with the direction of the light other than a right angle. The central reflection is only seen where the vein is running in a plane perpendicular to the line of sight. In a part at which the vein slopes, there is no central reflection, and so the vein appears darker, because in the centre the colour of the blood is seen as it is at the sides. Therefore, whenever there is the appearance of swelling of the disc, observe most carefully the veins as they pass on the sides of the affected area, and notice whether there is loss of the central reflection. If there is considerable swelling there is sure to be this loss. In this case it is scarcely perceptible, and therefore the amount of swelling is slight. The other method by which the amount of swelling can be easily estimated ought to be familiar to you, because it is of such wide application to every condition in which there is a difference in the level of objects in the fundus; as, for instance, to the depth and shape of the "cup." It is that which is called "parallactic," because, by an almost presumptuous use of a word which has a wide and important meaning to designate an exceedingly small subject, the word which describes the means of ascertaining the millions of miles of distance of the earth from other planets is applied to ascertain whether one object in the fundus of the eye is a millimetre in front of another. It depends simply upon the fact that the relative position of two objects is different if you change your point of view. In the case of the ophthalmoscope,—if, in the direct method, you move your head a little to one side and back again; if in the indirect method, you move your lens in the same way,—you see a change in the relative position of the objects. In astronomy it is necessary to wait until the other side of the earth's orbit is reached to observe the change in the relative position, but the principle is essentially the same. Never omit an opportunity of practising it in ophthalmoscopic work. Whenever you look at even a normal disc, move the lens a little, or move your head in the direct method, and you will be surprised how the relative change of position of objects at the edge of the disc and at the bottom, demonstrates to you

their relative distance almost as perfectly as if you were looking through a stereoscope.

The great fact of optic neuritis is the blurring of the edge of the disc in consequence of increase of tissue in front of it. The opacity of the tissue prevents the normal reflection of that which is behind, which can pass without difficulty through the quite translucent normal structure. Remember that the blurring always begins upon one side. You know that the nerve fibres which come from the optic disc, and radiate on the retina, come off on the nasal side, and above and below, but scarcely or not at all on the temporal side, towards the yellow spot, in spite of the fact that the yellow spot is the centre of vision. Hence it is that, at that part of the disc, the structures are absent, the nerve fibres, and the tissue between them that is the chief seat of inflammation, present the first visible signs of neuritis. When it is intense and considerable, there is sufficient tissue, even where it is least, to permit inflammation to invade the whole periphery of the papilla; but when it is slight the changes are limited to the side on which they are first seen. This is well seen in the figure before you and in the patient from whom it has been drawn. I am anxious that you should carefully observe it. The inflammatory change obscures the edge above and below, and on the whole nasal side, while it is absent on the temporal side.

In normal states there is always such transparency of the structures in front of the edge of the disc as to enable you to see it, even where the nerve fibres are most abundant. The first effect of neuritis is to increase the obscuration by making the tissue, which is over the edge, less translucent, and so to cause the apparent "blurring." Thus indistinctness of the edge is the first manifestation of the process. But the edge may be indistinct from another condition. You may have much difficulty in seeing it because the outer part of the disc is so red as to be of the same tint as the choroid. It is through this that the mistake is often made to which I have referred, the mistake of thinking that a disc that is simply and naturally red, is inflamed. This important distinction, however, is not difficult. The rule that you should fix in your minds is this. Let me first explain its reason. When you look with the indirect method, the degree of magnification is low, the illumination wide, and focus deep; when you look with the direct

method, there is a much higher degree of magnification, and a much more limited illumination and shallower focus. It is so with the microscope; a low power affords a considerable depth of focus; with a high power you have a very shallow range of focus. Hence, when you use the ophthalmoscope by the direct method, with its high magnification, you have a very shallow layer of focus compared with that which is afforded by the lower power of the indirect method. Hence it follows that if there is anything in front of the edge of the disc, obscuring it, when you examine by the direct method this layer is focussed, and not that which is behind it; the latter is not perceived, partly as it is not in focus, partly as that which is before it is in focus and so is distinct. With the indirect method, however, a lower magnification, a deeper focus, and a greater illumination enable us to see distinctly the tissue in front of the edge of the disc and also that which is behind this tissue, the edge of the disc itself. With the direct method, real obscuration is greater than it is with the indirect. But if the edge is indistinct from resemblance in tint of the disc to the adjacent choroid, when the eye is examined with the direct method the edge of the disc is *more* distinctly seen. Thus there follows this most important rule, that, if you think there is commencing neuritis, when the appearances are more natural with the direct method than with the indirect, it is normal; they are more morbid when there is more obscuration with the direct method. This is a rule of great practical importance; you may trust it, and if you endeavour to apply it you will soon discover its value.

I intended to describe to you the symptoms which the patient presents, but I think it is not worth while to do so, because the case is one in which a precise diagnosis is impossible, and in which the process of attempting to make a diagnosis is not of such conspicuous use as to justify me in diverting your thoughts and overcrowding to-day's memory. She has slight weakness on one side, a history of some giddiness, and of some headache of varying degree. A little weakness is thought to exist in the external recti, but it is not conspicuous. There is no nystagmus, and if it were not for the optic neuritis I think we should scarcely be inclined to consider that there was any organic disease. It is one of those cases in which there may be a small slowly growing tumour in some situation, or a tumour which is stationary, or increasing only at intervals as a tubercular tumour may do. But the symptoms themselves do not indicate tumour, nor does the optic neuritis.

The last statement may surprise you. The chief and first significance of optic neuritis is the presence of organic disease. If you can exclude a blood-state, or constitutional condition, you may feel sure that there is organic change within the skull or orbit, such a change as could be recognized almost always with the naked eye, and always at any rate with the microscope. The organic brain disease causing neuritis is of an irritative character. Irritation is the one element with which we can connect the process of neuritis, but the questions of mechanism and causation I must deal with upon another occasion. The point I wish now especially to press upon you is that, when neuritis is due to intracranial organic disease there is a relation between the course of the morbid process in the brain and the visible inflammation within the eye. Hence, neuritis is important, not only for diagnosis, but for prognosis. Many times I have known the commencing subsidence of neuritis to be the first indication of the commencing subsidence of a morbid process in the brain, and I have known the persistence of neuritis to be the indication of persistence of disease in the brain, of which every other effect, for the time being, had passed away. In each instance, and each class of instances, the indications have been absolutely verified. Further, chronicity indicates chronicity : chronicity of the neuritis, chronicity of the process. The converse is not always true. A chronic process in the brain, going on slowly, sometimes causes an acute development of neuritis. Doubtless the exception is only apparent; some acute consequence of a chronic growth causes the acute neuritis. A rapidly growing tumour never causes a chronic neuritis.

Neuritis is, however, sometimes so chronic that you may watch for month after month, and observe no change, and then, if there is organic disease, you can be certain that it is of a most chronic character. That is the case with this patient. I say, "If you can be sure of organic disease," because I have seen cases of slight optic neuritis of extreme chronicity in which, although there was no constitutional condition to explain it, I could not be sure of organic disease. Indeed, I have sometimes felt sure there was no organic disease. Those cases at present are a mystery. In a few cases, during a year or

two years, I could see no change in the aspect of the discs, and in one of them I came to the conclusion that it was a congenital condition. But conclusions, Gentlemen, seldom merit their name. Most conclusions should be regarded as beginnings, and not as endings, if you can pardon the paradox; it happened that five years afterwards I saw this patient, and every morbid appearance had passed away. It is possible that hypermetropia aids in the development of the condition. But all the patients, to whom I refer, presented evidence of functional disturbance of the brain, and I think there are cases in which very chronic optic neuritis is due to the combined influence of what we call "functional" brain affection and hypermetropia. How these causes act is still a very open question. But in none of those cases was the process sufficiently active to lead to extravasation of blood, and although in this patient, during the four months she has been under observation, no change has been perceptible in the optic neuritis, there have occurred the small extravasations of blood, which show an activity that is definite and seems to take the case out of the category of those that I have just been referring to. (Here again I have to guard the statement.) I say "seems," because the features suggest the question whether the occurrence of the extravasations may not be specially related to the peculiar arrangement of the tissues and vessels, and therefore whether it has so much meaning as we can generally ascribe to them. Remember that exceptional facts are often related, and that an unusual condition may be the cause of an unusual feature—that the preceding, congenital state here may be the reason why there are such hæmorrhages as are scarcely ever observed in neuritis that is at once so slight in degree and so extremely chronic in its course.

It will be more useful for you to carry away, instead of a confused perception of the possible meaning of the other symptoms presented by the patient, a clear perception of this one fact. There are cases of optic neuritis in which the process has not its common significance. There are cases in which it does not even help us to say that organic disease is present, in which it leaves the diagnostic problem very much as it would be without the neuritis. It should, indeed, invariably have one effect upon our minds. Like many other symptoms, in many other morbid states of various kind and in every part, it should make us more watchful, more careful to maintain our observation, and more ready to give due weight to any unequivocal symptom that occurs,—remembering ever that symptoms that are unequivocal alone may be decisive when combined.

THE PROGNOSIS OF LATERAL CURVATURE OF THE SPINE.

BY

A. H. TUBBY, M.S. Lond., F.R.C.S. Eng.,

Surgeon to Out-Patients, Evelina Hospital for Sick Children; Surgeon to the National Orthopædic Hospital.

THE question most frequently put to the surgeon when he sees cases of scoliosis is, "Will the patient grow out of it?" If the case be one of definite scoliosis, an answer in the negative may at once be given. Should only lateral deviation exist, the answer may run that with due care and attention to treatment great improvement will be effected, and the deformity will probably disappear. But in no instance can it be said that the patient has grown out of either lateral deviation or scoliosis. In some cases, the deformity is arrested without treatment after a longer or shorter interval, but some distortion persists through life.

Bradford and Lovett remark, "Two errors in prognosis are common. First, that the disease is of the most serious nature; second, that it is a trivial affection, and will be out-grown by the patient." Both statements contain just such a measure of partial truth as to render them misleading, and must arise from a want of careful appreciation of the factors at work in individual cases. In the succeeding remarks it is my endeavour to reiterate such points as may be useful in forming an approximately correct opinion. I say "approximately" as two kinds of cases defy calculation. That class which in spite of careful treatment is perverse from the first, and stubbornly refusing to yield, pursues a steadily down-hill course. Fortunately these are exceptional. The other class of cases is that in which the disease becomes arrested spontaneously in spite of the fears of the surgeon.

The elements of prognosis may be discussed conveniently under several headings.

The Cause.—Rhachitic deformities present many

difficulties in treatment. A scoliosis beginning so early in life is liable to be influenced for the worse by other factors which come into play when growth commences, and the disturbed bodily equilibrium persists with increasing force through all the years of growth. Additional difficulties arise from the tender age and small frame of the patients, rendering it difficult to fit apparatus. Scoliosis arising from perverted muscular action, especially in girls and at the age of puberty, require much caution in the expression of an opinion. Cases in which heredity is observed do not respond to treatment so well as those which are free from any such suspicion. Curvatures following empyema and pleurisy are always unfavourable for treatment, while those arising in connection with nasal obstruction and post-nasal growths are equally favourable.

The Age of Onset.—The later the commencement the more favourable may be opinion as to the ultimate condition. Thus, cases arising during the second dentition are likely to present less deformity in later life than those which begin in the first dentition. Similarly, those which commence between the ages of 16 and 20 rarely assume so severe a form as those which begin at puberty. More especially so if the former are due to preventible causes, such as bad attitude in sitting or static causes. Then, too, the later it appears, the nearer is the approach of the period when spontaneous arrest takes place, viz., at the cessation of growth. Repeated pregnancies when scoliosis has been quiescent for some years, frequently cause increase of the curvature.

The Sex.—Taken as a whole, in girls the deformity assumes a more severe and rapid form than in boys, owing to their want of muscular development, and the feeble health often entailed by disorders of menstruation. But some of the severest cases I have seen, have been present in boys, probably due to unsuitable occupations.

The Condition of the Bodily Health. This is undoubtedly one of the most important factors. Persistent anæmia and chlorosis may neutralize all efforts to obtain successful results. Similarly dysmenorrhœa and menorrhagia, from which so many female scoliotics suffer, by their exhausting effects prevent that steady attention to the improvement of muscular action and more perfect maintenance of equilibrium so essential to effect improvement. Girls in whom ill-health is tinged with hysteria are the worst subjects of all.

Many curvatures in town-bred people improve comparatively rapidly when the patient is sent into the country, and when the appetite and general tone are thereby bettered. If the health remain good all through, there is considerable hope of dealing successfully with a curvature of moderate severity, provided that no fixation is present.

The Probable Rate of Growth of the Patient. —Patients with long narrow yielding spines, which may be moulded into almost any shape, are bad subjects when scoliosis sets in. The probable rate of growth may be estimated by noting the patient's height, and comparing it with the average for that age ; the height of the parents should also be observed, and Galton's conclusions be remembered. These may be summed up in the following statement: "The deviation in height of the child from the average, will probably be two-thirds of the total deviation of the parents from the normal." This must hold good, otherwise the race would become all giants or all dwarfs.

The Occupation—Obviously, during the period of adolescence, persistence in the occupation which has induced the scoliosis can have but one effect. Unfortunately, in the case of the hard-worked eldest daughter of a large family amongst the poorer classes, it is one thing to advise rest, and another thing for the patient to be able to carry it out. It was mentioned above that some of the worst cases of scoliosis are seen in boys. This arises from the nature of their occupation, such as the case of a boy who carried a hod of bricks up a ladder from the age of 14 to 16 years; or that of a grocer's boy, who at the same time of life was employed lifting heavy weights. When growth is completed, occupations are far less likely to increase the curvature, unless they are too fatiguing, or there be a failure of the general health and strength.

The Site of Curvature.—Lumbar curves are less favourable for treatment than are dorso-lumbar or dorsal. Curvatures high up, cervical and cervico-dorsal are particularly troublesome, owing to the difficulty of making any efficient pressure upon the vertebræ through the ribs; while the natural kyphosis in the upper dorsal region facilitates increase of the scoliosis. Primary curvatures to the left in the dorsal region are more lasting and more liable to increase than are right-sided curves. In the latter case the greater use of the right hand is a considerable factor in the production, and much may be hoped for if this habit be discontinued.

The Form of the Curve.—It has been pointed out that the long C-curve, mainly a single one, is that particular form of early scoliosis which is likely to assume a very severe aspect later. The flexibility of the spine in these cases, which allows it to be so readily moulded, is an element which gives great opportunities to the surgeon ; which, at the same time, if the case go untreated, allows the spine to settle down into very marked deformity, and that not slowly. I have known such a C-curve to develop into marked scoliosis with right-sided dorsal and left lumbar curve in less than ten weeks.

While large single curves are more easily straightened under favourable treatment, multiple curvatures of *equal* length are likely to be spontaneously arrested, especially if growth be nearly completed.

The Amount of Existing Curvature.—Attention should be paid not so much to the amount of displacement of the vertebral spines, but rather to the rotation as evidenced by the altered shape of the ribs and chest. Cases of " flat-back " associated with scoliosis are more grave than those with kyphosis. In the former case much more rotation exists than in the latter. Still graver are those cases in which the normal antero-posterior curves are reversed.

The Stage of the Curvature.—So long as there is decrease of the deformity on suspension, some improvement will be attained. If the spine is rigid and fixed, the most that may be promised is that treatment will prevent the condition becoming worse, but no amelioration can be looked for.

The Treatment.—The essential point is that it must be begun early. Treatment well directed gives good, almost brilliant results in cases which may be termed of the first degree. Complete cure is exceptional, and rapidity of improvement must not be expected, despite the opinion of some authorities.

A few words may be expressed on the time taken for a scoliosis of marked degree to be developed. Fisher,* in writing on the " Progress of Scoliosis," says, "that from the first stage . . . . in a few weeks, one or the other of the conditions which have been described as illustrating the three types of lateral curvature is developed." Undoubtedly the cases which develop so rapidly are those with a long upper C-shaped curve. In one case I saw the change occurred in ten weeks. In rickets I

have seen kyphosis converted into very marked scoliosis in less than three months. When the curves are short, and in the dorso-lumbar region, the development is slower than when they are entirely dorsal and of considerable length.

AFFECTIONS OF THE VULVA :

BEFORE PUBERTY, DURING MENSTRUAL LIFE, AND AFTER THE MENOPAUSE.

A Post-Graduate Lecture delivered at Charing Cross Hospital on Thursday, Oct. 25, 1894, by

AMAND ROUTH, M.D., B.S., M.R.C.P.

GENTLEMEN,—The vulva is situated at one of the mucous orifices of the body where skin gradually changes into mucous membrane, and where all the changes to which transitional epithelium is liable may be met with.

Anatomically the vulva may be said to consist of the labia majora, one on each side (which are covered with hair, and join superiorly to form the mons veneris), and all the superficial structures lying between them, bounded above by the *clitoris* and below by the *posterior fourchette* or anterior margin of the perineum.

In this space, covered by squamous epithelium and richly supplied by mucous and sebaceous crypts and glands, are many areas of importance. Above is the *triangular vestibule*, bounded by the *labia minora*, each of which divides into two to embrace the *clitoris*, which forms the apex of the triangle, whose base is the anterior margin of the roof of the vagina. Just above this base line is the opening of the *urethra*, with the orifices of *Skene's duct*, just inside it. In some rare cases, minute orifices have been found immediately below the urethra,—probably the anterior termination of *Gartner's ducts*, which are more or less persistent remnants of the Wolffian ducts, coming from the Parovarium in the Broad Ligament. These ducts run along in the Broad Ligament, outside the uterus, and sometimes get into the muscle of the uterus and then into the sides and roof of the vagina, and have been traced just below the urethra.

Posterior to the vestibule is the vaginal orifice, *practically* closed in infancy by a more or less perfect circle of hymen.

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* Ashurst's Inter. Encyclo. of Surgery, vol. vi. p. 1066.

Posterior to this again is the *fossa navicularis*, lying between the posterior portions of the labia majora, and bounded in front by the hymen and behind by the posterior fourchette.

The important ducts of the *glands of Bartholini* open on each side and rather above the true limits of this fossa, the ducts being just behind the hymeneal fold at the position where the fossa navicularis is merged into the general internal surface of the labium majus on each side.

The *labia majora* are well padded with fat, and beneath them lies the vascular bulbous leech-shaped body, about an inch long, the *bulb of Kobelt*, which is a large plexus of veins with loose intervenous cellular and erectile tissue, enclosed in a fibrous membrane. It is here that varix, thrombus, and hæmatoma of the vulva occur.

Lymphatics are also freely present, and this vascular and lymphatic richness accounts for the readiness with which the vulva becomes inflamed.

Inserted into the upper portions of the connective tissue of the labia majora are the terminations of the *round ligament* of the uterus, which pass through the external abdominal ring, surrounded by a pouch of peritoneum, the canal of Nuck. This canal may be patent and permit a hernia to descend, or if, as usually happens, its communication with the peritoneal cavity be closed, it may become distended with a hydrocele of the round ligament, forming a swelling at the upper end of the labium majus.

The vulva is throughout life exposed to certain risks and outside influences, but it will be seen that its risks vary greatly according to the time of life of the patient, which I have divided, for convenience, into three stages.

1. Infancy and childhood up to puberty.
2. Puberty to the menopause.
3. After the menopause.

In a longer treatise these stages might be further usefully divided.

1. *Before Puberty.*

In infancy, and until puberty is nearly reached, the vulva is exposed mainly to such risks as exposure to cold, to absence of cleanliness, to the irritation of threadworms (though this was not credited by so careful an observer as the late Matthews Duncan), to constitutional affections, such as erysipelas, which may be associated with erysipelas of the navel soon after birth, in cases of puerperal erysipelas in the mother. Congenital

syphilis, struma, noma vulvæ, and the vulvitis which may complicate measles, scarlatina, and small-pox, may also be named.

It will, however, have been noticed by all present that it is very rare for any of these, even the most dangerous of them, to spread beyond the limits of the vulva and its surrounding connective tissue and lymphatics. The reason of this is twofold; firstly, because the vaginal mucous membrane and the other parts of the generative apparatus are, at this period, functionally inactive, and secondly, because the hymen forms a natural anatomical barrier to the upward spread of the disease, even though it be swollen or œdematous, as it often is.

Thus, in many bad cases of infantile vulvitis it can be shown that the vagina is healthy by carefully cleansing the vulva and vaginal orifice, and then, with the little finger in the rectum, cause the vaginal mucous membrane to bulge through the hymeneal opening, and it will be seen to be a pale pink as compared with the red, or sodden, or ulcerative state of the vulvar aspect of the hymen itself.

Of course, if violence has been used, as in some cases of infantile gonorrhœal vulvitis, vaginitis may accompany the vulvitis.

As a rule, however, the vulva alone is affected before puberty, and this makes the treatment much easier, for lotions can be very readily applied, and baths and fomentations become particularly useful.

2. *Between Puberty and the Menopause.*

In infancy the vulva is either carefully protected from cold by being well covered, or, if exposed, is exposed equally with the surrounding surface of buttocks and legs. Later on, children usually wear closed underclothing, and the vulva is less exposed than the legs themselves. At, or even before puberty, girls usually commence wearing open underclothing and the vulva is exposed to much cold and damp at a time when functional activity is starting, and when equal warmth is most essential. After puberty also, a woman leaves home more, and is apt to be exposed to cold updraughts in faulty closets, or to other similar inconveniences, which, especially if at the time menstruating, are serious and common causes of vulvitis, and even of consequent pelvic inflammation.

To realize the risks thus run, it will be needful

only briefly to refer to the changes which occur at puberty in the external organs of generation.

At puberty great mental and physical changes occur, and among the latter the changes which take place at the vulva are the most evident, and are an index of the still more important changes occurring along the rest of the genital tract.

These changes are—

1. A much closer nerve association between the generative and the general nervous systems, mainly through the increased development of the sympathetic pelvic ganglionic system.

2. The increase of vascular and lymphatic supply, regulated by ganglionic centres, and subject through their influence (it is believed) to certain monthly cycles; but even yet we have not the least idea of what causes these monthly cycles, or, indeed, what limits the months of gestation to nine, but it is supposed to be through some sympathetic influence.

3. Definite consequential development of the tissues, which in the vulva consist mainly of increased deposit of adipose tissue and increased activity of the secretory glands opening on to the different surfaces of the vulvar skin and mucous membrane.

The vulva now forms, not merely the outworks of a functionless interior, as in infancy, but constitutes the entrance to a series of canals, all of which are in a high state of functional activity, and along any one of which an inflammatory process may spread from the vulva by direct continuity of kindred tissue, involving organs whose inflammation may actually endanger life itself.

This is most marked in cases where the vagina, as well as the vulva, is primarily the seat of inflammation, especially if infective in type, as in gonorrhœa; but an ordinary vulvitis from exposure to cold, even in virgins, has often been known to be the starting-point of a pelvic peritonitis. I have seen it several times. The worst case of vulvitis and vaginitis that I ever saw, apart from gonorrhœa affection, was in a lady who was travelling from Scotland to London on a cold night. She was menstruating at the time, and was suddenly seized with diarrhœa, and had in consequence to retire rather frequently during the journey. The day was exceedingly cold, and the closet was a faulty one, the wind coming up with great violence, checking her menstruation, and causing acute vulvitis and vaginitis which extended

in that case into acute perimetritis, causing her very nearly to lose her life.

The vulva, therefore, is a portion of the body whose tissues are more vascular, more erectile, more sensitive, more secretive than any other mucous orifice of the body. It is a part, moreover, which, from its position and its functions, is liable to be exposed to cold air or to be contaminated by blood, urine, and possibly even, in dirty women, by fæcal matter; it is liable in the unhealthy, or careless, to be irritated by threadworms, pediculi, or dirt; to be irritated also by secretions from a diseased vagina, uterus, tubes, or bladder; liable to infection from gonorrhœa or syphilis; liable to injuries and excesses of a sexual nature; and finally, liable to injuries due to parturition.

Most of these rather alarming liabilities are peculiar to the period of life between puberty and the menopause, and a neglected vulvitis at this time of life may be a very serious matter.

I will enumerate a few of the complications which may ensue from a simple or a gonorrhœal vulvitis.

(a) Inflammation of Bartholini's glands.

(b) Inflammation spreading along the urinary apparatus.

(c) Inflammation spreading along the generative tract.

(a) Taking the more external parts first, and passing over such minor evils as œdema, ulcerations, etc., the most common complication is probably inflammation of the ducts and glands of Bartholini, whose position has been already indicated. These glands are embraced by the bulbocavernosus muscles, and the movement of these muscles and the tension of the gland greatly increase the pain of the vulvitis.

If a mere passive hypersecretion of these glands be excited, the transparent viscid secretion can be seen passing from the duct, along which a horsehair or very small probe can then be passed. If there be in addition catarrhal inflammation of the gland, pus or muco-pus comes away on pressure, or when the bulbo-cavernosus muscle acts.

If the duct be blocked at both ends a retention cyst is formed at the site of the duct, and if the duct be blocked at its outlet and the gland itself be hypersecreting, a retention cyst forms, which may reach a large size, and extend along the side of the vagina, burrowing deeply into the connective tissue.

At first it may be a simple retention cyst, but it may be inflammatory from the beginning, or may become so secondarily, and give rise to intense suffering.

Even if a catarrh alone result, and no cyst be formed, the red tender orifice of the duct can be seen for many months afterwards behind a fold of hymen, and is a common cause of a persistent dyspareunia and vaginismus.

(b) Another complication of vulvitis is urethritis, which almost invariably includes inflammation of the two ducts of Skene, which pass along the floor of its anterior third, and are sometimes evident at the everted edges of the inflamed urethral orifice.

The other urethral ducts and glands along its floor also may become inflamed, giving rise to the great swelling of the canal and the tenderness caused by pressure along it, through the vaginal roof.

Occasionally one of the urethral ducts or glands becomes a retention cyst, forming a swelling between the vagina and the urethra, and either inflames and suppurates, and opens into vagina or urethra, or bursts during parturition. If the opening have been into the urethra, a painful urethral abscess results. The urine trickles into the opening, setting up renewed irritation, and forms a constantly discharging abscess. The patient can get rid of this by pressing from the vagina, but unfortunately when she does this some of it is liable to get into the bladder, and then a cystitis is set up.

Usually, however, the shortness ($1\frac{1}{2}$ in.) and patency of the urethra, and its comparative freedom from lacunæ and glands, enable it to be kept well irrigated by the frequent micturition which results, and the inflammation does not long persist, nor does it often spread upward into the bladder and kidneys as in the male.

A neglected urethritis may, however, especially if kept up by a chronic gonorrhœal vulvitis, lead to a catarrh of the bladder, which is very difficult to cure. Urethritis may also be one of the indirect causes of vascular caruncle, granular urethritis, fissure of the neck of the bladder, and prolapse of its lining membrane.

Fissure of the neck of the bladder is the most painful condition, and its diagnosis is mainly arrived at by exclusion, because it is so difficult to see it unless one uses a cystoscope.

(c) The third group of complications of middle-life vulvitis is along the genital tract, which is the commonest way for inflammation to extend in females. If not concurrent, vaginitis usually follows the vulvitis, with formation sometimes of vaginal retention cysts from the blocking of one or more of its few gland-ducts, or distension of a persistent fœtal structure. Endocervicitis and inflammation of the vaginal portion succeed with small retention cysts, formed from blocking of the ducts of Naboth.

Endometritis, salpingitis, peritonitis, or ovaritis follow by direct continuity of tissue.

It is impossible to deal with these complications now which I have discussed in a previous lecture on vaginitis, but the main lesson to be learnt is to do nothing which can possibly carry up infection to a higher level of the genital canal by incautious examinations.

If the inflammation be purely vulvar, which is rare, avoid soiling the vagina. Inspection should be adopted in all cases of vulvitis, and if one finds that the vagina itself is healthy no attempt should be made to explore further. If the vagina be at fault and the endocervix apparently healthy, do not probe the cervix; if there be much endocervicitis, and no evidence of swelling or pain in the body of the uterus, avoid passing anything along through the natural anatomical and physiological barrier, the internal os; and if there be endometritis try to cure it before the barrier, the narrow passage between the uterine body and the free tube, is attacked, and salpingitis set up.

Much damage of this kind is done by the incautious use of the uterine sound, which should, in these days of improved manual and bi-manual examination, be hardly ever used, either for diagnosis or treatment.

Every one has heard of disaster and even death from infective or septic peritonitis following its use, and it is, of course, useless to clean the sound well if it has to pass through a septic or infective vagina en route to the uterus.

Personally I rarely use the sound, and when I do, I endeavour either to introduce it through a speculum or through a previously antiseptically cleansed vagina; and if I am in any doubt as to risk having been incurred, I subsequently pass up into the uterine cavity a Playfair's probe armed with wool, dipped in a 1 in 1000 corrosive sublimate solution, or in tincture of iodine.

No one would dream of dilating a uterus except under so-called strict antiseptic precautions, and yet similar precautions are rarely taken for the

passage of the sound where precisely identical organs are involved and similar risks run. Indeed, the danger may be greater with the casual use of the sound, because drainage is less complete than after free dilatation.

The poison is passed up into the uterine cavity, from which there is small chance of it coming out till the mischief is done.

The vulvitis may be simple or infective (gonorrhœal), or may deeply involve the subjacent connective tissue (phlegmonous), or may affect to an unusual degree the crypts and glands (follicular).

In addition to labial cysts the vulva may get attacked, between puberty and the menopause, by different forms of rashes, which may be eczematous, herpetic, pruriginous, or furuncular. The vulva is subject to early or late manifestations of acquired syphilis and to gonorrhœal warts.

It may be affected by lupus, especially the hypertrophic form, by tuberculosis, and by elephantiasis.

New growths, as epithelioma and scirrhus, are also not rare. We have already alluded to varix, thrombus, and hæmatoma.

3. *After the Menopause.*

This might be usefully divided into two groups, (1) climacteric, and (2) senility; but the third period of a woman's life is that of diminished sexual activity, with a tendency to retrograde changes of an atrophic character in the generative organs.

The vulva partakes of this process, and its adipose tissue gets gradually absorbed, whilst its glands cease to actively secrete, until, when senility arrives, the parts may be quite dry and shrivelled.

At or about the menopause a woman is inclined to get stout, and the folds of the skin are apt to get red and eczematous from irritative secretions, and the vulvar margins partake of this tendency.

A little later a woman seems predisposed to get *pruritus pudendi,* especially if she be gouty, or have very acid urine, and most especially if she be diabetic.

A pruritus at this age, with a coppery or purple rawness of the vulva, is often the first evidence of diabetes, and is usually associated with gout, and therefore a less serious form of diabetes than the early non-senile and non-gouty form of younger women, in whom this local evidence is not nearly so common.

One very rarely sees rawness of the vulva from any cause under the age of, say 45, but after that it becomes relatively very common.

Another important change to which elderly women are liable is *kraurosis vulvæ.*

I am not sure it is described in any English book of reference, and yet it is a common disease, and is in nearly every German book fully described.

This is an atrophic change, often associated with senile atrophy of the vagina, and sometimes with senile adhesive vaginitis.

It is accompanied by very severe pruritus, and the skin and mucous membrane have a dull white or glistening pearly appearance, due to degeneration of the superficial and deep epithelial cells, which here and there become detached, leaving shallow irritable ulcers, which greatly add to the discomfort of the patient.

In some of these cases the patient is in a dreadful state of annoyance and pain. While one part of the vulva is covered with small ulcers, the whole of the rest is white and intensely itching and irritating.

This whiteness may extend over the whole inner parts of the vulva and also to the perineum, even surrounding the anus.

In the vulva, kraurosis is always associated with a tendency to contraction of the vaginal orifice, with a liability to tear and split if coitus be attempted, and finally the orifice may become almost completely closed.

In this last stage, therefore, to recapitulate the main troubles to which the vulva is exposed, are rashes, mainly of an eczematous or pruriginous type, which often have a gouty or diabetic origin.

In addition to these, kraurosis vulvæ and carcinoma attack the vulva after the menopause. And, to make the list more complete, vascular caruncle of the urethra is very common.

(The author proposed to deal in a succeeding lecture with the treatment of the various affections, the etiology of which had now thus been dealt with.)

Tannigen.—Derivative of tannic acid not injuring the stomach. Yellowish-grey powder, soluble, inodorous, tasteless. Of value in subacute diarrhœa of phthisis and in chronic diarrhœa. Dose 3 to $7\frac{1}{2}$ grains usually sufficient, 46 to 60 grains daily producing no bad effect.

(Deut. Med. Woch.)

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

A Good Example of Bazin's Legs.

Amongst the patients who attended on Tuesday, November 20th, 1894, was a young woman, aged 19, tall, pale, and of very feeble circulation. She had been sent by Mr. Hurry Fenwick. Her hands were chilly, and showed a remarkable condition of dusky venous congestion. Both her legs, from ankle to knee, were covered with ulcers, many of which had livid edges and a grey surface, some being almost phagedænic. Together with the ulcers there were many livid, dusky patches, and many lichenoid papules and small pustules. The feet were quite exempt, and above the knees there were only a few indurated papules. On the backs of the upper arms there were a number of hard, red papules, of a lichenoid character. The patient said that the ulcers of the legs had been present only seven months, but the appearances suggested a much longer duration. There were no other evidences of struma, and the family history disclosed nothing that was noteworthy. The two legs were affected with almost similar severity.

Comments.—We recognize under the name of Bazin's legs an affection which occurs in connection with feeble circulation and scrofulous tendencies. Indurations and unhealthy ulcers occur on the legs from ankle to knee, which assume characters that, by the uninitiated, would certainly be regarded as conclusive evidences of syphilis. As such they used to be ranked until within the last few years. The differential diagnosis is very important, for we now feel no doubt whatever that they have nothing to do with syphilis. In the patient before us there is not the slightest reason to suspect syphilis, either inherited or acquired. She is a tall, overgrown girl, congenitally of feeble circulation, liable to chilblains, and whose strength has very probably been weakened by the menstrual function. It is precisely under such conditions that we see Bazin's legs. I have no doubt that the malady, when once it has begun, is an infective one, and that the original induration sheds into the blood certain elements which have the power of infecting other parts. Thus the disease soon becomes symmetrical as regards the legs, and we know quite well which parts will be the next to suffer. The disease will climb but very little above the knee, but it may appear on the backs of the upper arms. As to its pathological anatomy, in the best marked cases the first stage is always a subcutaneous induration, very possibly in a lymphatic trunk. Over this a dusky congestion of the skin occurs, and, lastly, the unhealthy ulcers which we have seen to-day. I have not been able in our patient to demonstrate the indurations, the inflammation of the skin being so extensive that they are concealed. Nor am I prepared to assert that these indurations are always observed, for in some cases, and especially in the later stages of the affection, lichenoid papules and pustules seem to be the first stage of the ulcers. It is, however, to be clearly recognized that Bazin's "indurated erythema of the scrofulous" is not in its best marked forms an infection beginning on the surface. The exemption of the feet is possibly to be explained by the protection afforded by the boots, for feebleness of circulation is a very important feature of causation. In this also we get an indication for treatment, for the warmth and protection afforded by Martin's rubber bandages is often of the greatest use. In the patient before us you will observe that, although as yet we have had no cold weather, she has already a dusky chilblain on one finger, and you have heard that she attributed the beginning of her ailment to "blood-poisoning" from a festering sore on one finger, caused by a slight injury. This expression, "blood-poisoning," conveys fairly well my idea as to the pathology of the affection. I do not suppose, however, that any poison is introduced from without, but rather that it is generated by the act of inflammation, in the tissues of a person in feeble health. The antecedents of Bazin's legs are, unquestionably, feeble circulation and scrofulous tendencies. In any person in whom these conditions meet, any slight injury may cause an inflammation which may "poison the blood."

A Case of Lupus Vulgaris with Scrofula.

The subject of this case was a man, aged 56 (sent by Dr. Alfred Eddowes), who in early life had had glandular abscesses in his neck, and more recently fistula *in ano*. One of his brothers

and a maternal aunt had died of consumption. The present interest of his case consisted in the existence of a large ill-margined patch on the left side of his neck, which was dry, of a brownish-yellow tint, and covered with exfoliating plates of epidermis. It did not weep nor was there any trace of ulceration or of tubercle. Its borders shelved off insensibly into the adjacent skin, and its whole appearance was very suggestive of the diagnosis of dry eczema. It was, however, in reality, as proved by the presence of scarring, a patch of lupus vulgaris in an exceedingly quiet condition (lupus exfoliativus). Under his chin on the other side of his neck, there was a smaller patch, which was destroying the hair of his beard and leaving a scar. Had this patch been present alone, everyone would have called it "Sycosis." The man gave a history of the patches having been present for fourteen years, and he had none elsewhere. He had never himself been suspected of being phthisical.

Comments.—This case is of interest, first, as illustrating the well-recognized connection between lupus and tuberculosis, and, next, on account of the peculiarities of form which the lupus has assumed. I am accustomed, as you know, to frequently insist upon the reality of hybrid diseases, and of the convenience of the use of double names. The term eczema-lupus may, I think, be suitably used when the appearances present would at first sight suggest eczema, and when there are or have been patches of eczema on other parts. So with the term "acne-lupus," where a lupus process attacks the pustules of acne. The lupus process probably depends upon the tubercle bacillus having been called into activity. It is very possible that this bacillus may be latent in the body for many years, perhaps from birth, and that it waits to manifest activity until a part receives some damage as regards its vitality. Is it, then, very improbable that a patch of eczema or a pustule of acne should now and then be attacked by it, and that these common skin diseases should stand in the condition of local predisponents? As regards the disease which we know as "sycosis," a pustular inflammation of hair follicles, which is infective, which spreads slowly, which disorganizes the part and leads to the formation of large glossy scars, I feel no doubt that it is in its nature very closely allied to lupus. This suggestion has been made by others as well as by myself. In the patient we have just seen, the patch in the man's beard must

present for anyone determined to make a verbal diagnosis a quite insoluble problem. I cannot conceive of any means by which it could be satisfactorily settled whether it ought to be called lupus or sycosis. My own impression is that it partakes of the nature of both maladies as regards alike its anatomy and its cause, and I think that clinical convenience would be served by giving it a double name.

A Case of Xerostomia.

The subject of this case was a thin, pale woman of 56, looking older. She had been sent from the London Hospital by Mr. Waren Tay. For ten years past she had been the subject of dry mouth without any intermission. She dated the malady from a severe illness, which was considered to be "inflammation of the bowels and congestion of the liver." On recovering, she found her mouth quite dry, and so it had remained ever since. The dryness involved not only the tongue, which was like that of a parrot, but her cheeks, lips, palate, and pharynx. No flow of saliva occurred during eating, and she had great difficulty in taking any kind of solid food. During the ten years her health had not failed in any very definite manner, but she said that she had recently got thin and weak. On one occasion, not long ago, she had had a severe attack of blood-vomiting.

Comments.—The patient before us affords a very good example of the condition known as Xerostomia. The case is exactly like others of the group. Indeed, it is a remarkable fact that since the first case which I brought before the Neurological Society, the accumulation of evidence, which has been considerable, has added nothing to our knowledge of the nature of the malady. In that case the patient was a woman of about the same age and of much the same appearance as the one now before us. In it, as in the present instance, the symptom of dry mouth persisted for many years without leading to any other form of disease. We conjectured then that the condition depended upon disorder of central parts of the nervous system, and demonstrated that there exists some centre from which it is possible to inhibit at once the salivary and buccal secretions. We can only urge the same hypothesis now. Whatever the affection of the nerve centre may be, it is clear that it is not infective or aggressive; for

case after case has been recorded in which, as in the present one, for ten years together a dry mouth has been the only inconvenience that the patient has experienced. None of the cases have been attended by dryness of the eyes or of the skin. It is a remarkable fact that the want of saliva, its entire absence indeed, does not appear in any appreciable degree to prejudice the patient's digestion. It interferes with comfort and enjoyment in eating, but there the matter seems, for the most part, to end. All the cases which have yet been recorded have, I believe, been in women, and mostly in those past middle life. It does not appear that the condition is subject to any remissions, or that, when once established, it is ever materially alleviated. Jaborandi, which was at one time recommended, is I believe of no real use. If given in sufficient doses to cause moistening of the mouth, it produces perspiration also, and makes the patient feel very weak.

Vaccinia Prurigo.

A little boy, nearly 2 years old, was sent by Dr. Fletcher, and afforded a good example of a vesicating pruriginous eruption, which had persisted ever since vaccination eighteen months ago. The child was not materially out of health, but was tormented by the occurrence of small isolated vesicles on various parts of the limbs and trunk, which, when scratched, became little festering sores. No obvious scars had, however, been left, nor was there any staining of the skin. The sores were isolated and not grouped, and the skin between them was quite healthy. They were most abundant on the limbs, and a few occurred also on the trunk. There were none across the loins or buttocks, [the parts constantly affected in the pruriginous lichen that results from flea-bites]. The mother's account was very definite. The child had been quite free from eruption until vaccination, and had come out about a week after vaccination in a general eruption, which looked like chicken-pox. From that time to the present, although better at times and worse at others, it had never been free. At first the eruption had consisted of little clear vesicles, which showed no tendency to fester, and it was only of late that the latter quality had been assumed. The vesicles had frequently formed on the soles of the feet.

In illustration of this case two portraits were produced showing similar eruptions, but in yet more severe forms. In one of these the sketch was taken only a few weeks after the vaccination, and the unhealed sores of the latter were still seen on the child's arm.

Comments.—The pruriginous eruptions of young children, to which the older dermatologists gave such names as strophulus, lichen pruriginosus, urticaria perstans, and the like, must in the present day be re-classified in connection with their causes. I hope I have succeeded in convincing you that some of them, at any rate, are induced simply by the bites of fleas and lice. We have had recently some very remarkable examples of these. In the present instance the cause is, however, wholly different. We have to deal in this patient with a persisting vaccinia eruption. It is, of course, not at all infrequent for an exanthem eruption to follow vaccination, but in the majority of cases it subsides quickly, and shows no tendency either to persist or to recur. In a small number of cases, however, due, no doubt, to some idiosyncracy on the part of the patient, the spots come out over and over again, and are attended by much pruriginous irritability. Precisely the same statements are true of varicella itself. Thus we have a vaccinia prurigo and a varicella prurigo, and of these the latter is by much the more common. They are both of them extremely intractable under treatment, and, in well marked cases, persist for a year or two, and it may be for longer. They always, however, so far as my experience goes, get well in the end, and leave the skin perfectly sound. I do not know of any treatment which does good other than attention to the general health and the use of soothing and healing ointments. It is very important not to allow the vesicles to degenerate into sores, or, in other words, to fester. The most careful attention, however, to local treatment will not cure the disease; it must wear itself out. In making a diagnosis between a flea-bite pruriginous eruption and one that has followed an exanthem, we must attend in part to the history of the case and in part to the localities affected. The limbs are usually most involved in the exanthem forms, whilst the trunk is the favourite site for flea-bites. Across the back, just below the loins, is the part specially preferred in the latter, and, as you have seen in the case before us, it may wholly escape in the former. In the cases following vaccination you will usually, as in the present instance, have a clear statement on the part of the parents that the

eruption made its appearance within a week or ten days of the vaccination; and this will often be strengthened by the addition that, in the first instance, it "looked like chicken-pox." In the chicken-pox cases there will usually be a history that some one—the doctor, the nurse, or the mother—suggested in the first instance that the eruption was chicken-pox, and this diagnosis was only abandoned because it did not go away. The soles of the feet are very commonly affected in the vaccination and chicken-pox cases, whereas they are scarcely ever bitten by fleas or lice. It is, perhaps, scarcely necessary to add that in these cases no suspicion attaches to the quality of the vaccine matter. The peculiarity is in the patient.

(A tolerably full account of this form of pruriginous eruption will be found in the author's "Clinical Lectures on Rare Diseases of the Skin." Vol. I.)

REVIEWS.

The Theory and Practice of Medicine. By F. T. ROBERTS. 9th Edition. (Lewis.)
Published at £1 1s.

To those who are familiar with this favourite of student days the appearance of a ninth edition will be very welcome. To criticise a work the worth and general appreciation of which are so thoroughly vouched for, would be almost an impertinence, and certainly we do not feel called upon to undertake the task. From the preface we quote Dr. Roberts' own words, "The present edition has been carefully revised throughout, and many parts have been re-written, while several subjects which have come into recent prominence are now discussed for the first time." We have looked through the book with especial reference to this statement, and can fully confirm it. Morvan's disease, the thyroid treatment of myxœdema, chapters xx. and xxi. on the circulatory system, all recur to us as illustrations of this confirmation.

Roberts' "Medicine" is still very widely read by students, and the more or less tabulated points in many diseases render it peculiarly suitable for their use, and we can give no higher praise to the present edition than to say that we think it will now be in greater demand than ever by all who wish to get a ready survey of diseases in a compact form. It is a large book (pp. 1152), but could hardly be smaller to contain, as it does, a complete system of medicine. Bristow and Taylor will have hard work to compete with Roberts as general text-books for students and practitioners of medicine.

Elements of Surgical Pathology. By AUGUSTUS J. PEPPER. (Cassell and Co.)
Published at 8s. 6d.

Pepper's "Pathology" is a book well known to students, and as it has now reached its 4th edition must be appreciated by a large number. Inflammation is a subject that one naturally turns to in a pathology as striking the key-note of the whole. Here we can say that the description of the process seems fully up to date in the mention of the modern views of that process, and the teaching on the subject can certainly lead no one into error. The illustrations are good, and the work as a whole is a satisfactory condensation of our present knowledge. We congratulate both author and publisher on the demand for this latest edition, though we do not like the divorce between pathology and the science of surgery. Their connection is too close and intimate to allow of independent study.

THERAPEUTICAL NOTE.

Quinine in Cholera.—Ten grains in powder, diffused through small quantity of water or in acid solution, at hourly intervals, until 20 to 40 grains have been given; afterward, *pro re nata*, the same dose at half-hourly intervals for sufficient time in collapsed or *foudroyant* cases; smaller doses, perhaps, at longer intervals, in choleraic diarrhœa. As other treatment, appliances for restoration of heat; saline hypodermoclyses to supply lacking serum to the blood; morphine hypodermatics to allay cramps and pain; enteroclyses of quinine in rare cases where remedy is vomited; calomel in small doses for subsequent enteritis or persistent diarrhœa.—(*New York Med. Journal.*)

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

ACUTE YELLOW ATROPHY OF THE LIVER.

Delivered at St. Thomas's Hospital, Oct. 29, 1894, by

SEYMOUR J. SHARKEY,

M.A., M.D. Oxon., F.R.C.P. Lond.,

Physician to the Hospital.

GENTLEMEN,—The subject which I have selected for to-day's lecture is that of acute yellow atrophy of the liver, one of the' rarest and most fatal diseases in this country. You will remember the following case, which was under my care quite recently :—

M. P., aged 21, servant, was admitted into St. Thomas's Hospital on September 19th last ; she was transferred to one of my beds in Charity Ward on October 3rd, and died on October 5th. For three weeks previous to admission she had suffered from a vaginal discharge ; this had been unaccompanied by any other symptoms for the first fortnight, but for a week she had had jaundice with clay-coloured stools, her food had produced discomfort in the abdomen, and her bowels had been constipated. When admitted, the skin and conjunctivæ were bright yellow, the urine contained bile, and there was tenderness in the region of the liver.

On September 26th, a week after admission, vomiting began to occur half an hour after food two or three times a day, and the bowels were obstinately constipated. From September 29th, vomiting increased in frequency, and almost everything was immediately rejected. She was sleepless, restless, and had a subnormal temperature.

On October 1st, a sudden elevation of temperature occurred—101° F.—and a motion passed was slightly streaked with blood.

On October 2nd, a diffuse, general, petechial rash appeared, not at all unlike the eruption of typhus ; there was a rigor, and the temperature reached 103° F., but fell by evening again below normal. There was an evident change for the

worse in the patient's condition ; she was very restless, and passed everything under her. Some streaks of bright blood were seen in the vomit, sordes had collected on the teeth, and the pulse had become rapid and feeble. The motions were slate-coloured.

On October 3rd, she came under my care. She was found to be deeply jaundiced, and all over the trunk and limbs cutaneous maculæ and petechiæ were present, while on the calves were diffuse subcutaneous hæmorrhages. She was extremely restless, the temperature had risen as high as 103° F., and she had had a rigor. She had also vomited, and the rejected matter was said to contain blood. Her cheeks were very congested, and there were beads of perspiration both on the face and over the sternum. The pulse was 160 and the respiration 38, but there was no evidence of disease within the chest.

The liver-dulness commenced at the fifth rib, continued as far as the seventh, and then was replaced by a zone of resonance as far as the costal margin ; below this, again, the liver-dulness could be made out if the hand was pressed firmly down into the abdomen and then percussed, and it seemed to extend downwards for about three fingers' breadth. The physical signs appeared to show that the liver extended vertically at least as far as in health, but that it had retired from the anterior surface of the body. The spleen was not felt. There was sordes on the teeth, the breath was offensive, the tongue dry and brown. The patient passed her urine unconsciously, so a specimen had to be drawn off by catheter. It was neutral, sp. gr. 1018, dark coloured, contained bile pigment, but no albumen or sugar. No leucine or tyrosine crystals were found either in the urine as passed, or after evaporation. Urea amounted to 5½ grs. per oz.

On October 4th, the general condition of the patient was much the same ; the temperature had varied between 103° F. and subnormal. The pulse was 154, the patient completely unconscious, and perspiring profusely. The temperature ran up to 108.6°, and the patient died early on the 5th. Dr. Box, the medical registrar, found that the urine, which had slowly evaporated on standing,

contained numerous large bundles of acicular crystals, presenting the ordinary appearances of tyrosine.

The post-mortem examination was performed by Dr. Hawkins ten hours after death. The right lung weighed 15 ozs., and the left lung 14 ozs. The heart weighed 11½ ozs., the liver 3 lbs. ½ oz., the kidneys 15¼ ozs., and the spleen 11 ozs., while the brain weighed 3 lbs. ¾ oz.

The body was rather fat. No eruption was visible after death, but all the tissues in the body were deeply bile-stained. On opening the abdomen, the liver was found to lie entirely under cover of the ribs. The organ was extremely flaccid, so that when it was held about the middle its two ends drooped on either side like a wet pancake. Its surface showed a dull yellowish colour for the most part, while small areas, particularly towards the thin free edge, were of the natural colour, or perhaps more purple than usual.

This difference in coloration was more marked on making sections of the liver. It was found that the greater part of the right lobe (perhaps ¾ths of its bulk) was of a dirty orange colour, was structureless in appearance, decidedly softer than it should be, and rather granular on a cut surface (something like a pneumonic lung in this respect).

The greater part of the left lobe, and a small part of the right, on the other hand, presented a purplish-brown colour, and though not showing normal hepatic structure to the naked eye, these parts were as firm and as smooth as usual on a cut surface. There was no hard line of demarcation between the parts of the liver thus differently coloured; the one merged gradually into the other, although here and there some islets of purplish-brown substance were left stranded in the orange.

The capsule of the liver was healthy, no peritonitis here or elsewhere. Portal vein healthy, containing a little post-mortem clot. The common bile-duct appeared at first sight to be somewhat dilated, but on opening it up there was found to be no source of obstruction in any part of its course. It contained a greyish turbid fluid with a trace of green in it, but no collection of mucus. The gall-bladder was not distended; it contained about ½ oz. of the same fluid as was found in the bile-duct; its walls were healthy.

Intestines showed no disease: the small gut was rather distended and nearly empty, containing only a little pasty clay-coloured material here and there.

Pleura and pericardium were healthy. Lungs healthy, though rather œdematous. The heart showed no disease: the valves and general structure were healthy: the right auricle was distended with dark clot. The kidneys were pale and slightly bile-stained. Cortex was much swollen: no old disease. The spleen was very large and firm, while the uterus and ovaries were perfectly healthy, as were also the suprarenal capsules. The brain and membranes showed no change, and there was no appearance of disease about any joint.

Microscopical examination showed that in some places the liver cells were completely destroyed, and simply consisted of a mass of granules which had escaped from the disintegrated cells, and in other parts the cells were diseased and filled with granules, but had not yet become disintegrated. Post-mortem examination then proved this to be a case of acute atrophy, notwithstanding that the liver was of normal dimensions. It must be remembered that our judgment as to whether a liver is atrophic or not must depend upon our knowledge of its previous size. In this case we have no knowledge as to the condition of the liver before the disease came on, but post-mortem examination showed that it was in a condition of acute atrophy.

Let me recapitulate the most important facts of the case.

1. Patient attacked with jaundice (catarrhal?) without serious symptoms, and following upon gonorrhœa.

2. Supervention of vomiting and constipation.

3. Restlessness and sleeplessness.

4. Sudden elevation of temperature.

5. Diffuse petechial rash with larger hæmorrhages, rigor, high temperature, sordes on teeth, etc. Rapid pulse, unconscious evacuations, hæmatemesis. Retirement of liver from anterior abdominal wall, absence of leucine and tyrosine from urine, diminution in the quantity of urea, finally, complete coma, hyperpyrexia and death.

The ordinary symptoms of acute yellow atrophy of liver are—

Jaundice, rapid diminution in the hepatic dulness, hæmorrhages, stupor, delirium, restlessness, convulsions, diminution of urea, chlorides, phosphates, etc., in urine, and presence of leucine and tyrosine.

It will be seen that the majority of these conditions were present in the case which I have described. The main difficulties in arriving at an immediate diagnosis of acute yellow atrophy were these:—

1. Such symptoms occur occasionally in hepatic disease where, on post-mortem examination, no acute atrophy is found, *e.g.*, in hypertrophic and in atrophic cirrhosis.

2. Such being the case it is necessary, in order to establish the diagnosis of acute yellow atrophy, that we should observe a rapid diminution in the size of the liver. But time is required for this, and the patient died very quickly. You must remember that while a large liver-dulness means a large liver, small liver-dulness does not necessarily mean a small liver. For when emphysema is present in the chest and distension of the intestines in the abdomen, the liver dulness may be obliterated, and yet at the post-mortem examination the organ may prove to be of normal dimensions.

Moreover, if a patient has enlargement of the liver before the acute atrophy sets in, the organ may be found of normal weight after death. In our case not only did I make out the liver to have about the normal vertical extent during life, but it proved post mortem to be of the natural weight.

3. No leucine or tyrosine were found in the urine, and though the urea was greatly diminished in amount, the urine contained a very considerable quantity, viz., 5 grs. to the oz.

Acute atrophy of the liver is a very rare disease. I saw most, if not all, patients who entered this hospital and died during twelve or fifteen years, but I only met with one other case in addition to that which I have already described.

The following are the notes of the observations which I made upon it at the time:—

On August 15th, 1878, when, as resident assistant physician, I was admitting cases into the hospital, a girl, A. W., aged 18, was brought up by her friends, who said that she had been living by herself, and that very little was known about her. For fourteen days she had suffered from indigestion, vomiting, and jaundice, and on August 14th she had been delirious and insensible, and had vomited blood. At 11.30 a.m., when I saw her and sent her into the ward under Dr. Murchison, she had marked jaundice, she was retching violently, and was delirious.

On his evening round the house physician made the following note:—

"Well nourished girl, face not flushed, lips and teeth a little dry, no sordes on them. Tongue dry and brown, bowels confined, takes food badly. No paralysis, conjunctivæ yellow, surface of body deeply jaundiced. Pupils equal and active. Lies on her back insensible, and cannot be roused. Vomits very frequently, and is very restless. Her face twitches, she throws her arms and legs about in a purposeless manner, and screams loudly. Vomiting seems to rouse her a little, but she soon relapses into insensibility again. What she vomits consists of coagula of milk and some dark brown matter. She has no difficulty in swallowing. The pulse is 84, and of fair strength; respirations 24. The abdominal walls move well, and there is no tenderness or dulness in the abdomen. The liver-dulness is very small, amounting to no more than a strip an inch in breadth on the level of the 6th rib. The spleen is not increased in size. No abnormal signs in lungs. Heart normal."

At 1 a.m. on the 16th I made the following note:—

"Abdomen rather flat; hepatic dulness much diminished, beginning at border of ribs in right nipple line, and extending a very small distance below them; below the ensiform cartilage there is no liver-dulness. The pulse is 84, regular. Pupils dilated, equal, and insensible to light. The tongue is dry, red, and raw; no sordes on lips. Jaundice well marked; much vomiting of 'coffee-ground' matter. Temperature subnormal. Passing urine under her. Probably acute atrophy of the liver."

At 8.30 the same morning the note was:—

"Pulse 144, respiration 64; comatose. Stertorous breathing comes on in paroxysms in which the arms get rigid."

At 10 a.m.:—"Condition in most respects the same. No eruption on skin; still vomiting. Pulse 150. Liver-dulness at most represented by a line of deficient resonance, about as broad as the finger, on the level of the 8th rib. Temperature has risen to 105.4° F., and there are mucous râles all over the chest."

Death occurred at 12.30, the temperature having steadily risen, and reaching 106.8° F., fifteen minutes after death.

The following are the observations I made on the urine, which was drawn off by catheter:—

"Urine high-coloured, slightly turbid, but after being left to settle, the upper half transparent, the

lower occupied by a thin flocculent deposit. Sp. gr. 1020.

On pouring the urine slowly down upon a layer of strong nitric acid, a very dark green ring appears at the junction of the fluids, and the rest of the urine assumes a pale grass-green tint and becomes turbid. The turbidity does not disappear on heating. On adding tincture of iodine to some more urine a green ring appears at the junction of the fluids. On testing for bile acids with sulphuric acid and sugar, no reaction is obtained.

On adding nitrate of silver to the urine a very scanty precipitate appears. The quantitative test for urea by means of a solution of hypobromite of soda, shows that it contains 1 per cent.

On microscopic examination of the precipitate, one sees a considerable number of cells, like those from the tubes of the kidney. Besides nuclei they contain oil globules and tufts of crystalline rods of a mahogany colour. Leucocytes are also seen, and a few hyaline casts containing renal epithelial cells, in which are oil globules and crystalline needles. Rosettes of mahogany-coloured needles are also seen free in the urine.

When the urine is evaporated concentrically striated oval and round bodies appear (leucine), and rosettes of acicular crystals of a pale straw colour (tyrosine).

The urine contains no sugar.

Another specimen tested on the 16th gives much the same results, but a copious precipitate is brought down by nitrate of silver."

Autopsy. The body was well nourished and deeply jaundiced. In the left groin, below Poupart's ligament, was an old linear scar, as though an abscess had been opened. The glands in its neighbourhood were somewhat enlarged.

The blood was dark, tarry, and imperfectly coagulated; a few ecchymoses in the fat about the heart; pleuræ normal. The lungs contained irregular black areas, apparently due to blood which had trickled down the bronchi.

The liver was exceedingly small, 25 ozs.; it was not seen at all below the ribs. Not more than an inch or so of the organ in the nipple line was not covered by the lungs or intestines. In the middle line of the abdomen the organ did not come into view at all. On removal it was found to be very flabby; the capsule was smooth, and was thrown into fine wrinkles when the organ was bent in various directions. On section it pre-

sented a bright orange surface, intersected by grey streaks and smudges, which followed the lines of the vessels, and were probably due to post-mortem changes. The gall-bladder contained thick, green, tenacious mucus, and its internal surface was much congested. There appeared to be no obstruction to the main bile ducts.

The duodenum was injected in patches. The stomach was filled with dark-brown "coffee-ground" matter.

There was nothing remarkable about the spleen. The cortex of the kidneys was of light orange colour, and was evidently swollen, standing out in marked contrast to the dark medullary part. Nothing abnormal about the uterus, but some old pelvic cellulitis and adhesions, with hæmorrhagic effusion into the latter. Both Fallopian tubes were greatly distended, and contained a thick brown matter resembling a mixture of blood and pus. Both ovaries were enlarged and contained abscesses.

The microscope showed complete granular destruction of the liver cells.

This case was one of the most typical that one could see; just such a case as is described in the text-books, the liver being reduced to only 25 ozs. Of course people die at various stages of the disease, and consequently the liver may vary in weight in different cases.

With regard to the usual clinical history of acute atrophy of the liver, we may say, first of all, that it comes on either very suddenly, without the patient being otherwise ill, or, as in the two cases just described, it commences with a preliminary illness, lasting generally from one or two weeks to two or three months, but the average is a few weeks, not months. After the disease has lasted this length of time, the toxæmic symptoms supervene. In the cases which come on suddenly the characteristic phenomena are at once evident, and are not preceded by the preliminary symptoms which occur in the second class of cases; and when once those phenomena do appear, the patient rarely lives more than five days. The age at which the disease is most frequent, is from 15 to 25; cases have been observed as young as 2½ years, and as old as 60 years; but it is generally regarded as a malady which attacks young people. It is far more common in women than in men, and pregnant women are more liable to it than those who are not in that condition. Syphilis has sometimes been supposed to be the cause for the

disease. Some cases have been preceded by great mental emotion; and we know that ordinary jaundice sometimes arises from mental emotion, but it is not necessarily followed by the symptoms of acute atrophy of the liver. As a matter of fact, no particular train of symptoms constantly precedes this disease.

Certain poisons produce a condition of the liver which is similar to that of acute general atrophy of the liver, notably phosphorus, and sometimes arsenic or antimony. I cannot now go into the differences between the morbid anatomy of the liver in these cases and in acute yellow atrophy; but in my opinion, if similar, it is not identical in the two cases.

Now as regards certain facts in the pathology of the disease. One of the most important things is the size of the liver, and one must remember that that will depend upon a variety of circumstances. It will depend, in the first place, upon the stage of the disease, because, as I have already pointed out, some cases die earlier and some later. One can notice at the bedside in cases where good opportunities are presented for observation, that the liver shrinks very rapidly; consequently, even a day more or less may make a great difference in its size. The stage of the disease, therefore, is a thing which influences the size of the liver as seen at the post-mortem examination. It has already been remarked that the left lobe of the liver shrinks more than the right; it is generally small, and even when not small, it is usually exceedingly flabby, and the capsule wrinkles when the organ is bent.

Generally the colour is not only yellow, as indicated by the name—yellow atrophy, but yellow variegated with red: the yellow appearing as the earlier stages of the disease, the red at the later. It is clear that the disease does not occur at the same time and equally all over the liver. Some portions of the liver may be comparatively healthy, while others are in an extreme stage of degeneration. One finds, upon examination, that the cells of the liver are excessively granular, and often contain fat; they are shrunken, indistinct, and finally disappear entirely, so that when a section of the liver is made, nothing but the remains of blood-vessels and the like may be observed, without any cells at all. This rapid disintegration of the liver cells is the characteristic of the disease. Under no other circumstances, except those of acute yellow atrophy of the liver, does one find

the cells completely disintegrated; the yellow appearance is produced by the cells before the disintegration is complete, while the red indicates that the disease has advanced so far that the cells have for the most part disappeared. The stools are clay-coloured — at any rate, at the last — because bile is not discharged into the alimentary canal.

Another important point is the condition of the kidneys, which always show swelling of the cortex, and the latter generally exhibits a yellowish orange tint; on microscopical examination one finds granular degeneration of the cells lining the tubes, a condition nearly always dependent upon some poison circulating in the blood. Fatty degeneration of the heart occurs not infrequently, together with petechiæ, ecchymoses, and larger hæmorrhages. The two first are such as may possibly lead to the suggestion of typhus. Jaundice is very rare in typhus, though it does sometimes occur, but the eruption is not at all unlike that of typhus. The reason that I at once came to the conclusion that this was not a case of typhus was that the elevation of temperature had only been present for about three days, whereas the rash, if it were typhus, would indicate a much later stage of the disease, as it does not generally come out till the fourth or fifth day.

With regard to the cause of the disease I am afraid I cannot enlighten you. It appears probable that the destruction of the liver cells is due to a poison, but of that poison we really know nothing.

A very large proportion of these cases occur in women, and pregnant women; you have remarked, no doubt, that each of these patients had something abnormal in the genital tract: one was suffering from, or had just recently suffered from, gonorrhœa, while the other had a disease of the Fallopian tubes, which might very easily have given rise to the absorption of some poison. That there is some connection between the liver and pregnancy, or the subsequent processes connected with it, is shown by the fact that fatty degeneration of liver cells occurs in suckling women.

If little is known about the pathology of acute atrophy of the liver, next to nothing is known about the treatment. No treatment can build up the liver cells that have been destroyed; the condition is necessarily fatal. It has been stated, it is true, that some cases have recovered, but

while I cannot deny this, it is still open to doubt whether they were cases of acute atrophy of the liver at all. In a case in which only a portion of the liver had been destroyed, it is of course conceivable that the disease might be arrested. Once it has reached an advanced stage, however, no treatment that is known can arrest it. All that can be done is to treat the case on general physiological principles. Try as far as you can to rectify any of the physiological processes which are going wrong; endeavour to prevent vomiting; give as much good food as possible, and keep the bowels open, etc.

It is quite possible that you may never meet with a case of acute yellow atrophy of the liver in your practice, but if you do, I am sorry to say that I cannot tell you any plan of treatment which is likely to rescue the patient from speedy death.

SOME POINTS IN THE ETIOLOGY AND TREATMENT OF ACQUIRED FLAT FOOT.

BY

T. H. OPENSHAW, M.B., M.S., F.R.C.S.,

Assistant-Surgeon and Lecturer on Anatomy at the London Hospital.

THE importance of this subject and the frequency with which we are called upon to treat these cases, may be judged from the following statistics, and must be my excuse for this paper.

I find it on record that of 1444 cases of deformity occurring in 17619 surgical out-patients at the Munich Hospital, 338 were cases of flat foot. In other words, in every hundred cases of deformity, 23 were cases of flat foot, or 2 per cent. of all surgical patients. Flat foot affects the male sex twice as often as the female (they have more standing, and their occupation is usually more laborious); it is bilateral twice as frequently as unilateral (the weight of the body acts on each leg equally); the majority of the cases occur between the ages of 16 and 25, that is, during adolescence.

Four definite forms of acquired flat foot are commonly met with: the traumatic, paralytic, ricketty, and the static. The traumatic (after

Pott's fracture, for instance) occurs in 5 per cent. of all cases; the paralytic (*e.g.*, after infantile paralysis) in 3 per cent.; the ricketty (where the bones are presumably softened, genu valgum or other sign of rickets co-existing) in 3 per cent.; and, fourthly, the static in 90 per cent. The importance of the latter form is obvious.

Although the weight of the body plays some part in the development of flat foot in every form, yet the static, which arises as the result of long standing or weight carrying during adolescence, is *par excellence* the form in which the influence of the body weight is most uncomplicated, and therefore is best adapted for study. It is to this form more especially that I wish to direct attention.

In order to obtain a clear idea of its mode of onset, a brief review of the normal structure of the foot is necessary.

The foot was formerly described as consisting of one vaulted arch, and Lorenz was the first to point out that the bones of the foot are definitely separable into two longitudinal groups which, in the articulated state, form two arches: an inner arch comprising the astragalus, scaphoid, three cuneiforms, and three inner metatarsals; and an outer arch, consisting of the os calcis, cuboid, and two outer metatarsal bones. The outer arch touches the ground with the head of the fifth metatarsal in front, and the tuberosity of the os calcis behind. It must be remembered, however, that in the normal foot this arch is supported throughout its entire length by the soft structures of the sole. The inner arch, on the other hand, touches the ground in front at the head of the first metatarsal bone, and rests behind upon the os calcis, which is therefore described by some as being common to the two arches. Its apex is situated at the astragalo-scaphoid joint. This arch is supported by ligaments, especially the inferior calcaneo-scaphoid, and by the tibialis anticus muscle above and the tibialis posticus and flexor longus pollicis and digitorum muscles below. It forms the greater part of the vault of the foot, and is normally unsupported by the ground. Its posterior extremity, the astragalus, rests upon the os calcis at a point in front of the tubercles, and the inner arch therefore is partially carried by the outer arch.

Lorenz teaches that flat foot is due to a flattening of the outer arch, and to the inner arch slipping off the outer. This theory is, I think, not wholly correct, for it must not be forgotten that, although

in the unarticulated state, the bones of the foot seem naturally to fall into two groups, yet that the adjacent bones forming the two arches are very firmly articulated together, and that in this way very strong and very elastic transverse arches are formed. Thus, the five metatarsal bones form one transverse arch, the three cuneiforms with cuboid form a second, the scaphoid and cuboid a third. The sole of the foot may therefore be described as presenting a vaulted arch, supported behind by the tuberosities of the os calcis, externally by the soft parts of the foot, and the base and head of the fifth metatarsal bone, in front by the transverse arch formed by the five metatarsal bones; internally, by the head of the first metatarsal bone; whilst on the inner side posteriorly, it is unsupported, except by ligaments and muscles.

Let us now consider how the weight of the body impinges upon the foot. When, with uplifted toes, we balance ourselves upon the heels, the weight is transmitted directly through the astragalus and tuberosities of the os calcis to the ground. When the foot, therefore, rests flat upon the ground and the body is held erect, its weight will fall in front of the tuberosities, and the more the body is inclined forwards the more anteriorly the weight strikes the foot, until finally it rests directly over the anterior transverse metatarsal arch and is transmitted to the ground through the two extremities of this arch. As the body moves forwards and backwards, therefore, the course of the greatest weight upon the foot may be roughly indicated by an antero-posterior line. At any single point in this line the body weight will be distributed not only forwards and backwards, but laterally also; that is, externally to the outer arch which practically rests upon the ground, and internally to the internal plantar arch. It follows, therefore, that the nearer to the outer arch this line is placed, the more will be the weight carried by the outer and the less by the inner arch, and vice versâ. It is obvious also that during the passage of the weight of the body along this antero-posterior line the strain upon the inner arch will gradually increase to a maximum, and will then diminish. It will be greatest when the body is inclined slightly forwards.

Von Meyer and Beely have demonstrated the fact that this line in the normal foot passes through the second or third metatarsal bones. They assert that the first and fourth and fifth metatarsal bones are useful only for balancing purposes, and may be removed without impairing the utility of the foot, and that when the third metatarsal bone is alone removed the supporting power of the foot is more or less destroyed. This fact, however, does not prove that the line always passes through a definite toe, but merely shows how extremely important is the transverse arch of the foot, for the third metatarsal bone is the key-stone of this arch, which is, of course, destroyed by its removal.

All weight carried by the foot will, in a greater or less degree, be transmitted forwards, and will ultimately fall upon some point in this transverse arch; its destruction therefore, will, of necessity, considerably impair the power of the foot to support weight.

So long as the patient stands upon one foot only the above-mentioned antero-posterior line passes through the third toe; but when both feet are used it will pass much nearer to, if not actually through, the great toe, as von Meyer has pointed out. When standing upon both feet, with the body slightly bent forwards, the body weight will strike the centre of the internal plantar arch itself, and will thence be transmitted forwards to the great toe, backwards to the heel, and outwards to the outer arch in each foot. The inner arch is, as above mentioned, prevented from collapsing solely by muscles and ligaments, and upon them, therefore, there will be the greatest strain, when the body and feet are in the above position.

The further apart the feet are held, or the more turned outwards the feet are, the more will this antero-posterior line be displaced inwards, until it fails to strike the great toe, but crosses the inner border of the foot. The line described by the weight of the body when walking with the foot turned well outwards, in fact becomes a curved one, commencing at the heel, running forwards and then turning inwards across the inner margin of the foot.

From what has been said it is not difficult to see why prolonged standing on both feet, with the body slightly bent forwards and the feet turned out, should be the attitude, of all others, which will most quickly produce a bulging of the inner portion of the arch and border of the foot.

It remains now briefly to enumerate the principal changes which take place in the position of the bones, etc. The outer arch becomes more or less flattened, and in exaggerated cases the cuboid and metatarsals are bent upwards at the calcaneo-cuboid joint, so that the arch be-

comes concave upwards instead of downwards. The external malleolus is blunted and in exaggerated forms, its flattened apex articulates with the anterior process of the os calcis. The os calcis is not only somewhat rotated forwards so that the facets for the astragalus become more vertical, but it undergoes eversion also, and the astragalus is thereby tilted inwards.

The astragalus is driven downwards and forwards, and even with a normal amount of superjacent pressure undergoes a certain amount of inward rotation upon a vertical axis. This rotation is much increased when the foot is turned out and the feet are held apart. In consequence of this rotation the head of the bone presses directly against and thus produces a bulging of the inner border of the foot. The astragalus also becomes so twisted between the malleoli that only the back part of its upper surface is in contact with the tibia, and at this point only is the cartilage fully preserved.

The scaphoid is displaced forwards slightly, but it chiefly rotates round the head of the astragalus owing to the dense and unyielding external and inferior calcaneo-scaphoid ligaments. In exaggerated forms of flat foot the scaphoid may even rest upon the upper and outer part of the neck of the astragalus. The cuneiforms and metatarsals are displaced forwards, slightly outwards, and lastly upwards also in severe cases. The front part of the foot, therefore, is displaced forwards, abducted, and elevated as a whole.

The inner border is elongated and bulges inwards, and the head of the astragalus can be distinctly seen and felt below and in front of the internal malleolus. The inferior calcaneo-scaphoid and all the plantar ligaments, although slightly elongated, are usually thicker than normal. In long-standing cases the articular cartilage remains normal only at the spots where the bones are in apposition; in other parts of the articular surfaces it is thinned, or becomes fibrillated, or is destroyed.

The following are some of the theories which have been held to account for the onset of flat foot.

Stromeyer attributed the condition to atony of the plantar fascia and ligaments. Hueter to abnormal growth of the joints. Reismann to muscular contraction; but neither of these theories accord with anatomical facts. Henke believes that the origin of the condition is due to fatigue of the calf muscles, and particularly of the tibialis posticus, which passes directly beneath the centre of the arch. He omits to attribute any effect to the tibialis anticus muscle. Lorenz, on the other hand, cannot believe that the effect is due to muscular fatigue alone. He admits that the muscles are capable of supporting the arch, but cannot think that upon them alone devolves the duty of supporting this arch continuously. I agree with him in thinking that muscular failure is a most important factor in the causation, but I consider also that in the normal foot flattening of the arch is prevented by an interlocking of the bones and by the ligaments which connect them together. Hoffa believes not only in the existence of muscular failure, but of an inherent tendency to bony softening which he says is necessary before flat foot can develop, "for only a certain percentage of people who have to stand long hours or who carry weights develop either flat foot or scoliosis."

The question remains, what is the cause of the muscular weakness? In connection with this question it is interesting to notice the frequent association of varicose veins in the lower extremity with flat foot. Doubtless the deeper muscular veins are simultaneously affected, and muscular weakness is not therefore an unnatural consequence. It is well known, too, that nurses are especially prone to flat foot, and this is attributed solely to the prolonged standing necessary. There is, I think, another and equally potent cause. In order that the step of the nurse may be noiseless, the ordinary boot is replaced by slippers with very low or no heels. When standing in slippers, the tibialis posticus is stretched the more the lower the heels, and the less the higher they are. This stretching leads to rapid fatigue, atrophy of the muscle and subsidence of the arch. In my opinion slippers should be discarded and the heels of boots for flat-footed people should be broad but reasonably high. The higher they are the more the weight of the foot in ordinary standing is thrown forward upon the transverse metatarsal arch, and the less tendency there will be for flat foot to develop. Another way of relieving the arch from pressure, which I may mention here, would be by having the sole of the boot very thick and the heel so low that the patient is practically balanced upon the heel. Boots so made are, however, very awkward to wear.

The indications for the treatment of acquired flat foot will best be obtained by remembering its mode of onset. It arises in young people, who

may or may not have a congenital predisposition to the affection, who have accustomed themselves to a gait with the toes turned outwards, or who are obliged to stand for many hours daily, and do so with the feet turned out and the knees somewhat bent. It arises in others who have recently increased in body weight, or whose work consists in carrying weights. The superincumbent pressure overcomes the power which maintains the arch of the foot, and the astragalus is driven downwards, inwards, and forwards, and thus the inner border of the foot is bulged inwards and becomes convex.

The first precaution to be taken, therefore, is this: all patients who are obliged to stand long hours at work should be taught that the feet should be turned outwards as little as possible, and anyone with a tendency to flat foot should be careful to walk with the foot straight rather than turned outwards.

Secondly, acting upon the supposition that the bones may be abnormally soft, the general nutrition should be improved in every possible way. Arsenic and phosphorus which, as we know, possess bone-forming properties, may be administered, the former in the form of pills, the latter in conjunction with cod liver oil.

Thirdly, remembering that the sinking of the arch of the foot is primarily due to a relaxation of muscles in consequence of fatigue our aim should be to strengthen these as much as possible. The best way to do this is by the use of massage and gymnastic exercises. The muscles of the sole of the foot and deep muscles of the leg, the tibialis posticus muscle more especially, should be massaged at least once daily for ten minutes.

The exercises appropriate for patients with flat feet have been excellently tabulated by Ellis and Roth, and are as follows :—

1. The patient with the feet parallel, the toes pointing straight in front of him, should practise raising and lowering the heels. He should practise raising himself as much as possible on the tips of the toes.

2. The patient should stand with the tips of the great toes in apposition and the heels apart, the angle formed by the feet being a right angle. With the feet thus placed he raises and lowers the heels, which should be kept as widely apart as possible during the movement.

3. With the toes still turned inwards and the heels separated, the patient should practise raising and lowering the heels, and flexing the knees at the word of command. 1. Raise heels. 2. Flex knees. 3. Extend knees. 4. Lower heels.

4. The patient sits with back supported, and keeping the knee extended, should practise circumduction of the foot, inverting and adducting it to the fullest possible extent.

5. Passive manipulative obstructed movements are called in to aid in strengthening the adductors (supinators) of the foot. The patient should try to adduct the foot whilst the surgeon opposes the movement; and secondly, the patient should endeavour to maintain the foot adducted whilst the surgeon forcibly abducts it.

6. Lastly, the patient should practise standing and walking with the inner border of the foot raised.

It is of the utmost importance to impress upon the patients the necessity of cultivating elasticity in the gait. The whole flat sole should not strike the ground simultaneously. The heel should be the first part of the foot to do so, and from it gradually the whole foot. By heel and toe walking thus performed with a good stride and with the feet parallel to each other, the muscles are exercised at every step, and much benefit will be derived.

If long standing is quite unavoidable the position of the feet should frequently be changed. It is advisable, also, that such patients should raise themselves on tip-toe frequently and invert the foot as much as possible from time to time. The importance of this must be emphatically laid down, and the patients must be taught that cure can be materially facilitated by their own efforts.

The above exercises, conscientiously carried out, will be found of the utmost benefit, and are, in my opinion, absolutely indispensable for the cure of flat foot without operation.

Fourthly, an endeavour should be made to remedy the valgus position of the foot, and then to maintain the amount of correction so attained. This may sometimes be done by gentle or forcible manipulation alone, or it may be necessary to keep the patient at rest in bed for a few days, or to administer an anæsthetic and to perform tenotomy of the tense tendons. When an improved position of the foot has been obtained (and it is not inadvisable that the valgus should be slightly overcorrected and the foot made to assume a position of slight varus) some appropriate support must be provided for the sole in order to

maintain the correction and thus afford time for the regeneration of the normal arch of the foot. Three methods of obtaining such support are at our command, viz.: 1. The valgus shoe; 2. the valgus pad; and 3. the valgus sole plate.

1. Of the valgus shoe there are two slightly different forms in use. In both of them, however, the object aimed at is to transfer the weight of the body to the outer part of the sole by converting the ordinary sole of the boot into an oblique plane. This is effected by increasing the thickness of the heel and sole on the inner side. They differ in that the heel in Beely's shoe is not only thickened on the inner side, but is broadened from before back on that side, and thus is said the better to compel the inversion of the foot.

There is no doubt that well-made valgus shoes are really efficacious. They are, however, somewhat clumsy and very conspicuous. If not well made also the patient soon treads upon the outer side of the upper leather, and the shoe becomes rapidly worn out and useless.

2. The valgus pad is made of india rubber, cork, or steel. It should be, but is seldom, shaped to the arch of the foot. It should always be fastened in position upon the inner half of the sole of the boot. For cases of paralytic valgus or where the valgus is combined with inward projection of the internal malleolus, a valgus pad may be ordered, and must be inserted into the sole of boots fitted with outside leg irons and a valgus T strap as made by Ernst. For all ordinary cases of acquired flat foot, however, the best method of supporting the arch will be by means of the valgus sole plate.

3. The valgus sole plate is made of 19 gauge steel plate or aluminium bronze. The essentials of a perfect plate are: first, that it should present an oblique plane to the sole, in order slightly to invert the foot; secondly, that it should support the whole surface of the sole, so that the weight of the body may be evenly distributed. It should exactly correspond, therefore, to the arch of the foot when normal, should slope gradually from within outwards, and should reach from the heel to the root of the toes.

American surgeons have taught us how these sole plates ought really to be made, and I owe to my friend, Professor Gibney of New York, the knowledge of the following method which he adopts. A plaster of Paris cast is first taken of the foot, which must not be allowed to press on

the ground, but should sink slowly into a box containing the liquid plaster, in order that the arch may not become flattened out. A plaster model of the foot, with the arch more or less unflattened, will thus be obtained, and this should be reproduced in iron. An iron casting of this plaster foot is easily obtained, and upon it any number of steel or aluminium sole plates may be hammered out. It is not essential that the sole plate should cover the heel, but if so made it may be secured by a screw to the heel of the boot. The plate should extend in front to within half an inch of the crease at the root of the toes. It should be curved upwards laterally to fit the inner and outer borders of the foot, but care should be taken not to prolong this too far upwards on the inner side, or uncomfortable pressure upon the scaphoid during standing and walking will result. The sole plate so obtained, if of steel, may finally be plated with nickel to prevent rusting, or may be covered with leather, and may or may not be secured in the boot. When worn out, broken, or rusted, it is easily replaced by a new one made upon the same cast iron model.

Thus far we have supposed that the arch of the foot was not flattened so long as the foot did not rest upon the ground, and that the plaster and iron casts therefore had each a fairly normal arch. In some cases of severe flat foot, however, it is impossible fully to correct the arch of the foot, and in others it is impossible to obtain a cast of the foot with the arch normal, even though supported by means of a loop of bandage placed beneath the arch during the process. Under these circumstances other steps are necessary.

In the first place an impression of the sole of the foot should be taken upon smoked paper. This will give us an idea of the degree of the flat foot present, and will be useful, if varnished and preserved, for future reference. Plaster and iron models should then be made with the foot in the best position attainable, and a sole plate made therefrom. This may be worn for some months, provided that the arch is fairly well supported. At the end of a few months the cast iron model may be filed down slightly, so as to present an increased arch, and upon this a new sole plate should be made. In this way the arch of the sole plate, and therefore of the supported foot, may be gradually increased from time to time until the normal is attained.

That such a sole plate does give a uniform

support to the arch of the foot and elevates the inner border without at the same time causing the foot to slip outwards, is easily verified by a personal trial of such a plate. By the support it affords to the inner border of the foot, the displacement of the astragalus downwards and inwards is prevented, and its prompt effect in relieving pain is thus explained.

In young people and in slight cases the sole plate will alone be sufficient to cure the flat foot; in older people and more aggravated cases, however, it is absolutely necessary that all the above instructions as to exercises, massage, etc., should also be followed out precisely, if the result is to be successful.

In cases of rigid flat foot due to spasm of the extensors and peronei, the foot must first be rendered mobile. A few days' rest in bed, combined with hot fomentations, will in some cases suffice to effect this; in others, where the spasm is very marked, 10 minims of a 5 per cent. solution of cocaine may be injected into the astragalo-scaphoid joint; in others, tenotomy of the peronei and extensors may be performed. In the majority of the cases, however, forcible manipulation under an anæsthetic suffices, and is effectual without tenotomy. When the shrunken soft parts have been stretched and the foot has been brought into a position of more or less varus by means of forcible manipulation, a plaster of Paris case should be applied with the foot in a position of slight over-correction. This plaster case should not be allowed to remain on longer than three or four weeks, lest muscular atrophy result. At the end of four weeks the plaster is removed and a sole plate is substituted. He should be told to exercise daily with the feet parallel to each other, and massage and manipulations should be daily applied. The cure is usually complete in three to four months, but the patient should always wear the sole plate.

In exaggerated cases it is often necessary to divide the tendo Achillis, in order to remedy the rotation downwards of the os calcis and astragalus, and thereby facilitate the restitution of the arch by forcible manipulation.

The adoption of these sole plates renders all forms of apparatus obsolete. They are light, cheap, easily obtained, fit the foot, render the employment of special boots unnecessary, and last but most important, they are efficient.

In the very worst cases of flat foot, where it is impossible to effect any considerable correction and where the patient is unable to stand or walk for long together, recourse must be had to operation. These are many and various: excision of a wedge from the head and neck of the astragalus (Stokes); extirpation of astragalus (P. Vogt); extirpation of the scaphoid (Golding-Bird); resection of astragalo-scaphoid joint (Ogston); cuneiform osteotomy of astragalus and scaphoid (Schwartz); and the oblique division of the os calcis (Gleich). Each of these operations perhaps is especially suitable to one particular case, but the one which I have found most frequently called for, simplest to perform, and attended with the best results, is the operation introduced by Professor Ogston, which is performed as follows.

With the patient anæsthetised, and the foot bloodless, an incision an inch and a quarter in length is made along the lower and inner border of the foot, its centre corresponding to the astragalo-scaphoid joint, which in these cases is displaced somewhat forwards. All structures down to the bone are divided by this incision. The joint is found and is laid freely open by an incision somewhat less than an inch long through the skin and internal lateral ligament, running upwards perpendicularly to the first incision. The inner part of the head of the astragalus becomes visible through the incised ligament. This ligament is next detached with the periosteum from the surface of the scaphoid, by means of a rougine. A stout chisel half an inch broad and bevelled on one side is applied now to the head of the astragalus with the bevelled side away from it, and the cartilage, plus a thin layer of the subjacent cancellous bone, is removed. The chisel is then used to the scaphoid, and a similar layer of cartilage and bone removed. This is best done by using the chisel with the bevelled side towards the bone, or by the use of a chisel slightly curved on the flat. It is advisable to remove all the cartilage, and this should be done with as little injury as possible to the subjacent bone. Sufficient bone must be removed to leave a good arch when the foot is inverted and adducted.

When the position of the foot can be fully corrected, the tourniquet is released, the hæmorrhage arrested, the wound is washed out with 1 in 40 carbolic acid lotion and sutured with silkworm gut. A new sponge, iodoform, carbolic gauze, wool, and bandage are applied. The bones

need not be pegged together and no tube is necessary.

At the end of a week, the wound being healed, the sutures are removed, and under an anæsthetic the foot should be brought into perfect position and put up in plaster of Paris for about a month.

I have operated upon nine patients, in three of whom both feet were affected, with the best results. In two of the three bilateral cases I operated upon one foot, and wrenched and applied a plaster case to the other simultaneously. Both patients, however, subsequently begged to have the other foot operated upon. In two only of the twelve operations did the wounds fail to heal by first intention. In them the subsequent exudation was pent up beneath a plaster of Paris dressing, and on this account I have been led to discard the immediate application of plaster of Paris.

The results were excellent, the arch in all being firm and, as far as could be ascertained, bony. Needless to say, these patients presented aggravated forms of flat foot, and had undergone other treatment in some cases for years.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

Nov. 27, 1894, by

JONATHAN HUTCHINSON, F.R.S., LL.D.

A Series of Five Patients, Illustrating Interstitial Keratitis and Syphilitic Teeth.

THESE patients had been brought by Dr. W. W. Griffin, one of the clinical assistants at Moorfields, by the permission of Mr. Waren Tay and Mr. Lang. The first of them was a woman, aged 46, whose eyes had been left almost blind by permanent and dense opacities in both corneæ. She had a pair of very well characterised notched upper incisors; but, with the exception of her teeth and her eyes, there was nothing in her appearance to suggest the diagnosis of inherited syphilis. She was a stout, well-developed woman, looking older than her years, with a prominent nose, which had a remarkably narrow bridge. Her eyes had never suffered in the least until the age of 38, when she passed through a most severe attack of symmetrical

interstitial keratitis. Her ears had never suffered. She was the eldest in her family.

Comments.—I have known but very few cases in which interstitial keratitis has occurred so late in life as the age of 38, and very few indeed in which permanent opacities of such a dense character have been left, yet it would not appear that this patient suffered much during infancy. Rather the condition of her physiognomy would suggest that she almost wholly escaped. Had she had snuffles, the bridge of her nose would have been expanded, and, had she had any serious eruption, we should have seen traces of it in the condition of her skin. There are, however, absolutely none, nor do her teeth suggest that mercury was used in infancy, yet no one can doubt, looking at her upper incisor teeth and her eyes, that she is the subject of inherited taint. Surely we have before us a fact that should excite our wonder. That it should be possible for a taint to remain absolutely latent for thirty-eight years, during the whole of which period the patient avers that she enjoyed excellent health, and, at the end of that time, without any obvious exciting cause, evoke such severe and symmetrical inflammation of the eyes, is certainly a most remarkable phenomenon. Yet it is one which we encounter, with only little less definiteness, in many other cases of inherited syphilis. Of course, we are not to suppose that the virus of syphilis remains extant in these patients during these long periods. No one ever hears of such patients becoming sources of contagion to others, nor are they liable, with the rarest possible exceptions, to any forms of syphilitic outbreak, excepting inflammations of the eye and ear. These two sense-capsules are each liable to be attacked and, let us note, usually with the most exact symmetry, in patients who appear to be otherwise in excellent health. No disease of skin or mucous membrane is usually present. If anything happens, it will probably be an attack of phagedæna in the nose or palate. This however is rare, whilst inflammations of the corneæ and of the structures in the middle and internal ear are common. The problem of explanation is an extremely difficult one; but the fact is one which ought to make a deep impression on our minds, and by the aid of which we may probably elucidate some other pathogenetic difficulties.

Case 2.—The subject of this case was a girl, aged 21, of a somewhat suspicious, though ill-marked, physiognomy, and with notched and

shortened central upper incisors. Her tibiæ were bent forward and thickened. Her corneæ were in an early stage of keratitis, and one of them showed in perfection the well-known salmon patch.

Case 3.—In this case, a girl of 18 had screwdriver teeth, and in both corneæ the remains of an attack of keratitis, which was rapidly clearing up.

Case 4.—The subject of this case was a girl of 19, in whom, after a year's duration, the corneæ had almost wholly cleared. Her central incisors were notched and too short.

Case 5.—In this case a girl of 12, of somewhat delicate appearance, had slightly notched teeth, with keratitis in both eyes. Her mother stated that the first part of her family had been born quite healthy, and had remained so; then had occurred three still-births, and after them the patient.

Comments.—I am very much obliged to Mr. Tay and Mr. Lang for kindly enabling me to make use of this important series of cases. First, let me ask you to note that all five patients are females, and next that they are all the eldest in their families. This remark is not literally true of the fifth patient, but the facts disclosed in her case suggest that the father acquired syphilis subsequent to the birth of the first part of the family, and that she was the first to live after that event. I made great use of the fact that the subjects of interstitial keratitis are, in the great majority of cases, the eldest in the family, when I was engaged in the attempt to prove that this disease is a direct result of inherited taint. The cases which we have seen are especially valuable in their negative aspects. There is not one among them concerning whom you could say that the physiognomy of inherited syphilis was really well marked, and in one at least, not the slightest trace of peculiarity can be said to exist. I may ask you to note also respecting their teeth that, although in all of them I unhesitatingly pronounce that the upper central incisors are typically notched, yet none of them have shown us any defects in the other teeth. It is a great mistake to suppose that in inherited syphilis, the whole set of teeth is usually conspicuously malformed. Conspicuous malformations of the teeth are much more frequently due to some other cause rather than to syphilis. A true syphilitic malformation is one which needs to be looked for, and that too by skilled eyes. I wish we could

get rid of such absurd terms as "peg-top teeth" and some others which are in use, and that we could fix our attention upon the upper central incisors alone, for they only give us reliable information. Every one of the five cases which I have shown you has illustrated this point. The common cause of the other more conspicuous and more extensive malformations of the permanent teeth is, I believe, the use of mercury in early infancy, and from this it happens that pegged teeth, indented teeth, and teeth with the enamel extensively defective are often seen in the mouths of those who are the subjects of inherited taint. They are not, however, consequent on the taint itself, but on the remedy used for its cure. Respecting the patients whom we have seen this afternoon, I would suggest it as very probable that none of them suffered severely in infancy, and that none of them underwent a very prolonged treatment. None of them have shown, in any marked degree, the malformations due to the use of mercury. Respecting Case 1, I would feel almost certain that no infantile symptoms at all occurred. This suggestion raises the interesting inquiry as to whether many patients, who really inherit a taint, show no manifestations of it in infancy. I suspect a good many do not, whilst a still larger number suffer only very slightly. You will see that this has a very important bearing upon practical matters in several directions. The unfortunate accidents which have occurred in the communication of syphilis by vaccination, have usually been in cases where the vaccinifer showed no signs of taint. So again, in arguing out the connection which I believe to be invariable between inherited taint and interstitial keratitis, we must be quite prepared to meet with a certain number, like the first case seen this afternoon, in whom all history of infantile symptoms is wanting. It fortunately happens that in these cases we are almost invariably helped out of the difficulty by the malformation of the teeth.

We may just note that in none of our five cases has the patient been deaf, disease of the ear being, although by no means infrequent, not nearly so common as keratitis; nor with one exception has any of them shown any sign of periostitis of the tibiæ. None of them are the subjects of lupus or of any form of skin disease; but this is not exceptional, for, permit me to insist, nothing is less common than for the subjects of inherited syphilis to suffer from such ailments after the period of infancy.

A Very Peculiar Case of Lupus Marginatus.

The subject of this case, sent by Mr. Waren Tay from the London Hospital, was a woman aged 40. Her skin disease was of six years duration, and consisted in delicate rings, scattered over the face, very abruptly margined, and presenting minutely lichenoid borders. They varied in size from a three-penny bit to a shilling, and in some instances had joined, and thus lost their circular outline. All of them were pale in the centre and more or less definitely in a condition of scar. The scar was however extremely superficial and thin, and in some instances, it might have been disputed whether it was really present. The "lichenoid rings" consisted of minute papules not so big as pins' heads. These, although small, were very definite, and were not attended by any erythema. In this latter feature, the patches differed widely from the discs of lupus erythematosus. One of the largest patches was on the side of the nose, three others were grouped in the middle of the left cheek. There were probably altogether twenty or more. They were not arranged in very exact symmetry, and there were two or three on the right ear, which had no representatives on the other side. The patient said that all the patches were steadily, but very slowly, increasing in size, but gave her little or no trouble. The family history was distinctly one of tuberculosis. The patient herself, although looking tolerably healthy, said that she had several times been suspected of chest delicacy, and that several of her father's family and one of her own brothers had died of consumption.

In illustration of this case, a portrait was brought from the museum, which showed a precisely similar condition on the face of a boy. It had been published in Archives, vol. i., plates 13 and 14.

Comments.—I have no hesitation in saying, nor do I think that you have any in accepting the statement, that the portrait shown and the patient produced are examples of precisely the same disease. So far as I know, they are the only examples of it yet observed, but we have some approach to the formation of these delicate serpiginous rings in another form of chronic skin disease, which is illustrated in Archives, vol. i. plate 9. It may be better however, that we confine our attention to the two cases only. They are alike not only in their external appearances, but in all the clinical history. In both, the disease was slowly aggressive,

lasting through many years, and yet producing no deep destruction of parts. I have no hesitation in calling the disease lupus, although it is very different from any of the other recognized forms. It is probably nearer to lupus erythematosus than to lupus vulgaris; at any rate the multiplicity of the patches would suggest this, but on the other hand we must note that it is not symmetrical, and that it is wholly unattended by erythema. Its extremely superficial character is one of its most noteworthy features. You will observe that the history of tuberculosis in the patient's family is strong. There can be little doubt that the whole family of lupoid affections is in some sort of connection with the tubercle bacillus; and we may conjecture that the peculiarities displayed by the different forms in this family are due to the special parts of the skin attacked. It would appear to be the law that the morbid process keeps to the special tissue first invaded. Thus we have an erythema-lupus, which remains erythematous throughout; and a sebaceous lupus, which displays the same peculiarity. I should like, if my dermatological colleagues will allow me, to add a lymphatic lupus in which lymph vesicles are the most conspicuous feature. In all these, infectivity, destruction of the tissue involved and the formation of scar are the essential features; and in all we have good reason to believe that there is some association with scrofula and tuberculosis. To this group our present case adds another special form. The term "marginatus," which I applied to the first case is applicable simply to the abruptness of its outline, and to the entire absence of any implication of the adjacent skin. It is however, merely used for convenience sake, and were it not that the disease is so extremely rare, would probably soon be superseded by a better. We might, if it be preferred, know it by the name of its first subject, and call it Hilliard's lupus.

Ectropion of the Lower Lip.

This patient was a woman of middle age, the subject of tertiary syphilis. The whole of her lower lip was completely everted and much thickened, producing distressing disfigurement. The upper lip was quite thin. The poor woman, who had been sent by Dr. Sequeira, was the subject also of very extensive syphilitic lupus on her neck and chest.

Comments.—This is the second example of

ectropion of the lower lip which we have had before us during the last year. It is a rare deformity and a very disfiguring one. Of the other patient, we have preserved a portrait in the museum. Ectropion of the eyelid is very usually caused by a cicatrix of the cheek. It is not so with ectropion of the lower lip. The lip is pushed out by swelling, not pulled down by contraction. Its subjects are almost always those, like our patient, who have suffered from tertiary syphilis, and in whom sores are formed repeatedly on the inner surface of the lip. These sores being attended by swelling, the lip becomes the seat of solid œdema, and is everted. When once produced, I believe there is never any natural cure. The specifics for syphilis avail nothing. The only treatment is by operation. Either an elliptical flap from the inner surface of the lip must be excised, or a similar result must be obtained by the free use of the actual cautery. I have had very satisfactory results from the latter measure.

An Example of Bazin's Malady.

A rather stout, married woman, aged about 36, brought by Dr. Hillstead. She had been the subject of multiple strumous ulcers on the legs for two years, and many dusky swellings and scars were present. As usual the feet were quite exempt, and the morbid conditions did not go higher than the knees. The circulation was very feeble, and her hands were tumid and dusky. Two of the patient's brothers, and two of her sisters were reputed to have been the subjects of consumption.

Comments.—We have had so many cases recently illustrating the condition now known as Bazin's malady, that I will not repeat any general statements respecting it. One of the chief points of interest in the present case is, that it furnishes another illustration of the association of this disease with the tendency to tuberculosis. A liability in this direction, in association with feebleness of circulation, are its usual causes. I may perhaps venture a word or two as to the name which it is most convenient to use. Bazin himself calls it "The indurated erythema of the scrofulous"; a designation which is unfortunate, because it takes no cognisance of the most conspicuous feature, which is ulceration, and names one—the induration—by no means invariably present. We might perhaps speak of them as "scrofulous legs", or "strumous ulcers of the legs"; either of which terms

would attract attention to the real nature of the most prominent character of the disease, "Bazin's malady" is a little too high sounding; and "Bazin's legs" a shade too familiar. I expect however, that the last title, as being the easier to use and the more suggestive, will be the one to come into general employment. That they are essentially scrofulous, meaning by that term that they are closely allied to tuberculosis, no one can doubt. I may just mention in this connection, that I have seen this morning in private a most typical example of these legs in a young lady, who had, some years ago, scrofulous glands excised from her neck.

THERAPEUTICAL NOTES AND FORMULÆ.

Biliary Lithiasis.—Treatment at Hôtel-Dieu, Paris. Hepatic colic of spasmodic form in patient with nervous temperament:

R   Infus. orange flower ...   f ʒ33½
    Chlor. water ...   ...   f ʒj
    Pure neutral glycerin...   f ʒ33¾

Dessertspoonful every half-hour until pain has ceased. Injection of Oj infus. senna ʒiiss. Second injection of Oss decoction poppy heads. Gentle friction of right hypochondrium every hour with flannel dipped in following liniment:

    Bals. Fioravanti   ...   ʒiiss
    Chlor. ...   . f ʒv

Vichy or seltzer water with cherry syrup as drink, skim milk or vegetable soup as food.

Hepatic colic with biliary retention in arthritic diathesis: Full lukewarm bath for at least one hour, renewed if necessary. In interval of baths, application to right hypochondrium of flannel dipped in strong very hot infusion of chamomile, covering with oiled linen. Applications renewed at least every hour. Following internally:

R   Sod. salic. gr.7¾
    Bicarb. magnes.   ...   gr.7¾

To make one dose in azymic cachet. Two or three injections of infus. senna ʒi¼, water Oj, neutral glycerin 1 tablespoonful. Calomel, followed by infus. hops ʒ33½, syrup of poppies, chloroform water, glycerin, each ʒv. One teaspoonful every hour. Alkaline water mixed with milk as drink.—(FERRAND, *Gaz. des Hôpitaux.*)

Relieve suffering by chloroform and morphia internally, or chloroform alone by inhalalation if suffering be great. Give calomel gr.viij, aloes and soda each gr.iij, packed dry in one capsule ; or, powdered ipecac. gr.xxx–xl, in two doses, two hours apart. Four hours later, following mixture, tablespoonful every three hours :

℞	Pot. bitart.	...	...	ʒiv
	Magnes. sulph. ...		...	ʒj
	Syr. rhei. pot. co.		...	ʒij
	Aq. q. s. ...	...		ad ʒiv

After recovery from acute attack, one capsule of following after each meal :

℞	Ferri red.	...	)	each
	Quin. sulph.	...	)	gr.xxx
	Leptandrin	...	...	gr.xv
	Ext. nux vomica		...	gr.vij
	Aloin ...	...	...	gr.iij

M. et ft. caps. xxx.

(*Memphis Med. Monthly.*)

Nightsweats of Phthisis.—Dr. von Szekely warmly recommends the *combination* of tr. bellad. and arsenic as follows :

℞	Liq. fowleri ...	...	♏xlvj
	Tr. belladonnæ	...	♏xlvj
	Aq. laurocerasi	...	ʒvj

Sig. : 10 to 15 drops to be taken about 5 p.m., and a similar dose on going to bed.

He also has found cotoin useful to help the digestion.—(*Munch. Med. Woch.*)

Wounds (Reclus's Method of treating Crush of the Extremities). — Slightly anæsthetize the patient by giving several whiffs of chloroform, and carefully wash all the injured parts with water at a temperature of 131° or 140° F., using a spray of sufficient force to thoroughly penetrate the interstices of the wound, expel blood clots and detached pieces of flesh, as well as any foreign bodies. Spread upon pieces of aseptic tarlatan a layer of salve made as follows :

Salol	...	...	)	
Resorcin	...	...	}	of each ʒ3¼
Antipyrin	...	...	)	
Boric acid	...	...	...	ʒv
Iodoform	...	...	...	gr.xvss
Vaselin	...	...	...	ʒv

Press the tarlatan into all the interstices, covering the entire wound. Surround the member with absorbent cotton. Renew the dressing every five or ten days. When the crush has not been too great, this treatment will often enable the limb to be preserved, while, if amputation become necessary, it can be performed later when partial cicatrization has taken place, thus ensuring a stump well covered with soft parts.

(*Thèse. de Paris.*)

REVIEW.

Diseases of the Upper Respiratory Tract, Nos·, Pharynx, and Larynx. By Dr. P. WATSON WILLIAMS. (John Wright and Co., Bristol.)   *Published at 8s. 6d.*

We do not remember having read any book on a speciality in medicine that has given us such sincere pleasure as this work by Dr. Williams. It is exactly the book which we should have eagerly bought in our student days as introductory to the study of diseases of the region. From cover to cover it is filled with just the kind of knowledge which the beginner requires. Mouth breathing on pp. 3–5, the succeeding pages on general semeiology, difficulties in laryngoscopy on p. 18, have charmed us by the simple style and language of their description—nothing verbose, nothing deep, but just a happy expression as though the author were speaking to us. The pages remind us of nothing so much as our old classics by Watson, Todd, and Graves. The body of the work has scattered through it numerous tabular compendia of points in differential diagnosis, which make it invaluable to students who think and require their knowledge systematised. Another excellence in the work is the illustrations, of which there are five full-page coloured plates and 120 wood-cuts, almost all of which will be found of use. A good many of the wood-cuts are illustrations of instruments, but those which are of the various regions dealt with in the text bear the impress of being personal experiences of the author.

All who wish to acquire in the most pleasant manner some knowledge of the diseases of the larynx and nose we strongly advise to buy this book at once.

A POST-GRADUATE CLINICAL DEMONSTRATION,

Delivered at the Cleveland Street Sick Asylum on Nov. 8, 1894,

By T. CLIFFORD ALLBUTT, M.D., LL.D., F.R.C.P., F.R.S.,

Regius Professor of Medicine in the University of Cambridge.

GENTLEMEN,—*Case 1.*—This is a case which you would put down bluntly as "heart disease." In practice a large number of cases which are not put down as heart disease are heart disease in their tendency to death; that is to say, the tendency to death is by way of the heart. Such cases you have to treat as cases of heart disease, although they may primarily be cases of pneumonia, bronchitis, and things of that sort. On the other hand, the case before us, which you would describe primarily and definitely as heart disease, is not heart disease in the practical sense. If you will listen in the aortic region (as Dr. Hopkins has kindly made out for us), you will find there a loud, direct, systolic murmur; at the apex there is another very loud systolic murmur. Both these murmurs, as any of you may at your leisure observe, are very distinct and loud. An inexperienced student studying a case like this would say, "There are two very loud murmurs, which must signify a most alarming state of things —heart disease of a very grave kind." So far as the patient here is concerned, I do not think that the murmurs have much to do with his illness. Although there is a mitral regurgitant murmur, his heart is beating quite regularly; he has no enlargement of the liver, no congestion of the lungs, no dropsy of the legs. He could not have legs cleaner in the tendons, and there is not the smallest trace of œdema of the skin. A loud murmur is too often assumed to indicate a bad heart affection; but it may, on the contrary, often indicate a mild one. If the ventricle act strongly and drive the blood through a tiny chink, a loud murmur may be heard; whereas a weak ventricle and very incompetent valve may give rise to a very soft murmur or possibly none. This patient

has loud murmurs, both mitral and aortic, but probably very little mechanical disturbance in these respects.

The next point to which I would refer, is that a direct aortic murmur is usually said to be due to aortic stenosis. Stenosis, of course, means straitness; now, straitness of the aortic orifice does occur, giving rise to a direct murmur, and also to hypertrophy of the left ventricle. This state, however, is rare; one has to take some trouble to find examples. What, then, causes the murmur in the common run of cases? Not the narrowness, as a rule. You may suggest that it is due to roughness of the orifice or valves, but this is not the cause either. It is a very common mistake to suppose that murmurs are caused by the passage of the blood stream over rough surfaces of valves or walls, as water babbles when it goes over stones in a brook. But the circulation is a plenum—is quite full. Fluid cannot ripple inside closed tubes where there is no air and all is tight. A murmur is almost always caused by the passage of the blood from a narrow space to a wide one. This patient has probably a dilated aorta, and, when the ventricle contracts, the blood goes from a small cavity into a wider one, namely, into the dilated aorta. It is generally, then, true of murmurs that they are not due to roughness, but to passage of the blood from a smaller into a larger chamber, thereby producing certain eddies called "fluid veins"—a term we have no time now to discuss. You will then say, "If his malady is not the heart, what is it?" Take either arm, bend the elbow, and look at his brachial artery; you will see that the artery is thrown into large loops; it is longer than it ought to be. How comes that? It comes about because the artery is giving way under tension, longitudinal as well as transverse. The normal and ordinary tension by blood pressures tends thus to stretch the artery, and the artery, if it yield, must be thrown into coils in order that it may lie within the same space from the shoulder to the wrist.

Now, this patient is nominally but 59 years old, but that, of course, is nothing like his real age; the case being one of premature senility. In medicine we do not count the ages of people

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by the revolutions of the earth round the sun, but we measure them by the revolutions of their own morbid processes. He is, as a matter of fact, a very old man, he is a man, say, of 75 or 80 years of age, speaking physiologically, and this senility is his malady. If you take hold of his radial artery and rub the tip of your finger up and down it, you will find its coats full of patches of what we call atheroma; the artery is gritty, having roughnesses or ridges on it like those of ipecacuanha root. Again, the channel of the artery is so far narrowed by this change that the stream of blood in it is diminished. He has then a thickened elongated artery with a small blood stream, and more heart's labour is required in propulsion. Consequently, there are anæmia and defective function of every organ; there is want of muscular energy, the memory and mental faculties generally are failing, and he has attained at 59 what he ought not to have attained until 75, if at all.

Therefore, although the case is put down as heart disease, the state of his heart is so far not the cardinal point; the man is suffering rather from cardio-arterial than cardiac disease. We have not then, at present, to estimate the relation of the pressures in the various chambers, or to treat the heart with digitalis and things of that sort. The patient, however, does speak of some pain in the præcordial region.

Now, it is the commonest thing in the world for young people to come to us with some anxiety, saying that they have pain in the region of the heart, but in ninety-nine cases out of one hundred it is of no serious import; but when a man in later life, suffering from cardio-arterial degeneration, complains of pain in the præcordial region, we must look at it quite differently. I do not say that this patient has a pain which we should conveniently call "angina pectoris." Yet there are no positive lines of division between the case of a man who dies from the sudden cardiac failure, perhaps with no cardiac pain at all, which is one of the modes of angina pectoris, and that of a man like this who merely has tiresome quasi-dyspeptic pains in his præcordial region. I say there are no fixed lines of division between those extremes. We persist in talking about "types" and "typical cases" of angina pectoris, and the like; but if you will take my advice you will not often use that word "typical," for it draws you away from the consideration of these exceedingly

important transitional forms, and leads you to dwell upon the fancy sketches of the lecture room, and away from the infinite variety of the wards. A man past middle age may come to you saying that he has a bothering sort of pain, "indigestion" he may call it. I do not mean a spasmodic pain, or a pain which makes him turn ashy-grey in the face, or thrusts his sternum into his back bone. Nor may he have exactly a sense of impending death, or of syncope; though things may get a little "far off" and "woolly" for him. Yet, at times, and with such pains such a person is insecure. While præcordial pains, then, in young people are usually of trifling importance and do not signify heart disorder, in senile people the persistence of a sort of præcordial pain has often a very serious meaning, and they may die as suddenly with pain which they merely call dyspeptic, nay, which you also call dyspeptic, as if they had what you call "typical" angina pectoris. This patient has præcordial pain; he describes it as a very "nasty pain," and says that when it comes on he feels occasionally also some faintness, and things get rather misty before his eyes. If a patient with failing circulation says things are getting misty before his eyes or to seem "far off," it may mean that the circulation is failing and may fail fatally at any moment.

We will now turn to three interesting cases of disease of the nervous system.

This patient is suffering from a complaint which, as you know, was named by Erb "spastic paraplegia." The second case is primarily one of phthisis, but he is brought under our notice because he has also peripheral or multiple neuritis. The third patient is suffering from that disease which it is best to call "tabes dorsalis." Locomotor ataxy is but a symptom, and a symptom common to more than one disease. Therefore it is not a good name for the disease before us; nor again is "posterior sclerosis," for such cases may present symptoms in the optic nerve, ocular motor symptoms, ophthalmoplegias of sorts, and vago-accessory symptoms (gastric crises, and things of that kind), which are not directly due to sclerosis of the posterior columns alone. It is therefore better to take a meaningless name like that of "tabes dorsalis," which does not commit us to anything. Let us now take, first, this man with spastic paraplegia. I dare say several of you notice something wrong about his feet; he has not got a "dropped" foot; the neuritic patient

in the next bed has, as you see, a dropped foot, but here you see the feet are not dropped. There is a little disposition to get the heels inwards, to bow the legs, and to get up a little bit of talipes equinus, due to the pulling up of the tendo achillis. It is not the foot which is dropped, but the heel which is drawn up. If you handle either tendo achillis you will find it very tight. The next thing you will notice is that he has "scissors palsy," his legs, when abducted, tend to clip together like shears, because the abductors are in a state of intense spasm. Again, you see, I shall have to use no little force to bend the knee up to a right angle ; but when I get it beyond the middle point, the flexure is completed pretty easily. When I again extend the limb, as it passes the right angle, it will spring back sharply "like a clasp knife," as the phrase goes. Those are points which are probably familiar to you. I will now assume, for the moment, that there is no important muscular atrophy, though you may notice that the left leg is, in fact, decidedly thinner than the right. In many of these cases, however, the over-action seems to make the muscles even larger than usual. We will also assume that there are no disturbances of sensation, no anæsthesia or paræsthesia. The patient, as do many such patients, will tell you that he has a little numbness, and numbness in hands or feet is an expression which may be properly used by him, but it is one which is difficult to define, and in these cases does not signify much. I will now press up the toes a little, and you will see that ankle-clonus is produced. The foot is now vibrating like a large tuning fork. This symptom may occur in hysteria, perhaps not so well marked as this, but sufficiently to embarrass diagnosis. Yet in hysterical spasm you will find that the resistance is not one which gives way at a certain angle, and on extension again recoils in " clasp knife " fashion, and so on, but the state is rather one of permanent contraction which you cannot get over, or only by much force, and the infliction of much distress.

Still, as I have had good reason to remember, and as Dr. Buzzard has also shown, diagnosis may in such cases be very difficult.

If we now examine the fingers carefully, we shall find that they are a little curved or coned on the palmar aspect, and with a little care tendon reflexes can be obtained in the arm : they are not so well-marked as one might expect to find them, but I got them quite plainly this afternoon, and you see something of them now. Now spastic paraplegia

does not very often remain a very pure clinical type. I have not gone more closely into this case than is necessary for our present conversation, but I have suggested that the left leg is wasting, and I think it is probable that the disease is beginning to complicate itself with the muscular atrophies of Charcot. In so far as it does that, it ceases to be a pure case of spastic palsy which, as you all know, depends upon a chronic degeneration of the pyramidal tracts, in any part of their length. In the early "congenital" cases of young children, the tracts are probably invaded even so high as their cerebral terminations, say, by a cerebral hæmorrhage, which may injure the upper ends of these motor tracts. In adults the lower or spinal "side strings" are generally invaded, but the mischief does not tend to remain, so long limited to them, as does sclerosis to the posterior columns. In Erb's palsy, the process may tend to take in the posterior columns, but more commonly to take in the anterior horns, and so lead to atrophy of the muscular tissues as well as to spasm. Such, then, is its disposition to associate itself with other diseases.

Let us now inquire into causation. With regard to tabes and its congeners, a great many persons say at once that they are due to syphilitic infection This infection seems less active in causing spastic palsy than in causing tabes. From my own experience, I should say that syphilis is not a very marked feature in the causation of spastic paraplegia ; as we have said, it may arise from more ordinary kinds of irritation : for instance, it may arise in a hemiplegic form from the irritation of a cerebral hæmorrhage in adults. Of tabes on the other hand, syphilis is a very common cause indeed. Many teachers tell you it is the only cause, and that there is no tabes without previous syphilis. That I think is unquestionably too absolute a statement. It is difficult to carry one's personal convictions into other minds, but I am convinced that I have seen some cases in which it was absolutely impossible that this antecedent could have been present, at any rate in its acquired form Therefore, I say that in perhaps ten per cent. of tabes, you have to look for another cause.

Now, in tabetic persons, in addition to the usual presence of syphilitic antecedents, there is very commonly a neurotic inheritance also. You may have the neurotic inheritance determined by specific disease, or rarely, you may have it determined by some other factor, such as a blow or injury. Again, in a substantial number of chronic spinal

degenerations you will find a history of a fall or like injury. I remember very well such a case in a young man from the north of England; he had first, syphilis, then, some eight or ten years later, as he was going down some iron stairs, he slipped, and fell heavily upon the bottom of his back; spastic paraplegia appeared soon afterwards.

In the case before us, the same event is ound, for this patient tells us he had a fall of some twenty-five feet from a scaffold on to his buttocks. There is also a definite specific history in his case; the infection was forty years ago, and his fall was twenty-three years ago. Soon after the fall the patient began to suffer from his spinal disease. Among the causes of spinal degenerations I find that a neurotic bias by inheritance is rarely absent. Perhaps the next most active cause, especially in tabes, is syphilis, and not infrequently another exciting cause is found in a blow or a fall. This patient began with the symptom of fatigue—intermittent fatigue—which commonly ushers in spastic paraplegia. One day, after walking four miles, he felt "terribly weary," and his toes dragged; "his legs felt stiff, and his toes caught in the ground." After sitting down for a little while he was able to go on better, and so on. He has some shooting pains, but these pains are not so important as they are in cases of tabes. In spastic paraplegia, so long as they are simple, there is no disposition to bed-sores or joint affections. The patient shall now relate to you another important symptom. Q. "Will you tell us what happens in your sleep?" A. "I sleep the first part of the night, but I generally wake up about half past one or two with pain in my left leg. My leg shoots up, as you noticed it shot up just now when you lifted the right one. It was in pain all the time until you took it down again. After two o'clock I have to battle with my left leg to try and get it down. The last two mornings, when I have tried to sit up and wash myself, I have found that I cannot get on to my posterior, as the twitching up is so painful."

This "twitching up of the leg into the body," as patients often express it, may be an early and important symptom. In this patient there is also an exceedingly tight, hard, and rigid state of the muscles of the abdomen, which, I think, is directly associated with his malady. Although bladder and rectum troubles practically never exist in pure spastic paraplegia, he has some little defect of that kind, but, as I have said, his malady appears to be extending a little to other tracts. He says he has great difficulty with the muscles of his trunk, in putting himself in position to perform the calls of nature. This is not so much that the bladder and rectum themselves are at fault, but that he feels a difficulty in so arranging his body that they can act. This arrest may be due to the spastic state of the muscles of the abdomen.

Case 3.—We will now turn to a patient with multiple neuritis—*i.e.*, inflammation of the peripheral nerves of the legs, not including any spinal centre. In this case you have a dropped foot. If you feel his Achilles tendons you will find that they are quite slack—different from the previous case. It is not that his heels are drawn up, as in the last case, but that his toes fall. In this case there is no tendency to talipes equinus. You easily lift his foot into its natural position; but the moment you let go it drops. In this patient the peripheral neuritis is associated with pulmonary tuberculosis: I will cautiously suggest that it may have come on in consequence of this phthisis. Peripheral neuritis is frequently associated with tubercle. You may reply that one may commonly find phthisis superadded to any chronic disease which lowers the general vitality; but as a rule in these cases the tubercle comes first, and the neuritis afterwards. When people are ill and bedridden such a condition of the legs as this patient shows might well escape notice; it is latent; there is not necessarily any pain, or there may be some wandering pain. This patient has a little pain in the sciatic and other nerves, but one might easily attribute this to lying in bed, or the like. Thus, neuritis may probably be much more common in the latter stages of consumption than we suppose, and often escape notice. The invalid cannot stand, but this would not be surprising in the latter stages of consumption. It would not occur to a clinical clerk to test the legs minutely at this stage; the nurse would think nothing of them, and the malady would be passed over. Now, if this connection be more than accidental—I only throw it out as a suggestion—is it due to the actual presence of tubercle bacilli in the nerves of his legs? I do not know; nobody has found them there, and I cannot tell you. I think that they are probably not there. But the tubercle bacillus, like other bacilli, may discharge toxines into the blood, and I need not tell you that multiple neuritis is a common consequence of many toxines. The diphtheria

bacillus, for instance, does not travel about the body—it remains in the throat, and there generates its poisons, which travel about the body, and cause also peripheral neuritis and other mischief. I suggest, then, that the tubercle bacillus need not necessarily be in the nerves, but that it may, as does the local bacillus of tetanus, and as that of diphtheria, brew these toxines, which are carried into the system, and that in this manner it may set up peripheral neuritis.

Case 4.—We now come to the case of "posterior sclerosis," which I said we should rather call tabes dorsalis. The general symptoms are familiar to you all; you know what they signify. They signify a loss of harmony between two sets of muscles, which I will but for the moment call the agent and antagonist muscles. The word "antagonist" is a bad one; no normal muscle is antagonistic to another. All the muscles concerned conspire together to the desired result, and I think, therefore, we muddle ourselves by speaking of "antagonist muscles." Let me impress upon you that the whole of the muscles of the arm, for instance, conspire to perform any one particular act the arm has to do. It is not that the flexors are ordered to beat the extensors, for instance, or *vice versâ;* but all being gathered together into a kind of group under what we call the "will," when a message goes to the proper centres, not one set or the other, but the whole of the muscles of the arm conspire to do this or that. In this patient, however, there is a loss of balance in the muscles, so that they do not conspire as they ought for the acts required. Now, perhaps, they do become antagonist, and extensors may prevent flexors from doing what they ought to help them to do. In this case, again, the patient had a bad fall; and as in the case of spastic paraplegia, there is here also marked evidence of neurotic inheritance. I need not tell you that a history of hereditary qualities is often hard to obtain, especially in hospital patients; yet, however clearly syphilis may be present, you will find, if you can get at the facts, that in those persons who know their family history well, a hereditary neurotic tendency is almost always co-operative, and is, perhaps, the main and fundamental thing. In this case an originally neurotic habit is associated with syphilis and with a severe fall. His gait became unsteady not very long after his fall; and we frequently find in syphilitic local manifestations of other parts that an injury, such as a

knock on the shin, may set up a greater activity of the specific malady, or arouse a dormant into an active form of it.

Now, let us again consider the effect of poisons upon the nerves. Do not suppose that when we speak of syphilitic disease of the posterior columns we mean that there is specific mischief in them; if you examine them under the microscope, you will find mere degeneration, and nothing else. Do not expect to find gummata or hyperplasias of the sort, but mere ordinary degeneration. If, on the supposition that the mischief is a specific inflammation, you give mercury or iodide of potassium, you will not do the smallest good. I believe that cases of syphilitic tabes which are supposed to have been cured by mercury and iodide of potassium have been cases of peripheral neuritis, and not of posterior sclerosis at all.

Nor would you expect thus to do any good in the case of mere degeneration. Then how is it that syphilis is regarded as by far the principal cause of tabes? I will repeat what I suggested to you about phthisis and neuritis, namely, that the bacillus may brew certain toxines in the body, which diffuse themselves and set up degenerating mischief, as do other poisons. You will not then be surprised in such cases to find that specific treatment fails to restore the vanished tissues.

I will not take you through all the ordinary symptoms of cases of tabes, but we will touch upon a few. The first little point I will mention is one which I think is not generally known; at least, I do not hear it mentioned, namely, that the pulse in tabes is nearly always, or very commonly, accelerated. After talking to this patient for ten minutes, and when he was perfectly quiet and still, I found that his pulse was 100. It is probably higher now that we are all about his bed.

Another characteristic of tabes is the tendency to tumble. The man falls down; he looks around, and wonders what on earth he has tripped over. Possibly he has tripped over some trifling little object, but very often there is no such object at all. However, he is convinced that he has tripped over something, but over what he cannot discover. Usually he tumbles rather heavily on his knees, elbows, or otherwise.

But I suppose on the whole the most useful of all the early indications is the loss of knee-jerk. In this patient there is, as is usual, a loss of knee-jerk—"knee-stop" as I often briefly call it. This symptom is almost universal, though not quite

universal. I have had a case under my observation lately, one in which there is now no patellar reflex, and in which tabes is but too well marked. Two or three months ago with care, and at one particular spot of the tendon, a little reflex could be obtained in both legs, but now that little has gone. Therefore knee-jerk is not always wholly absent from the outset. Another early character is the " Argyll-Robertson pupil "—the pupil which contracts on convergence, but not on light. I often call this the " light-stop " for brevity's sake. This patient has the light-stop, his pupils are not affected by light but by convergence only. Myosis, another very important early character, is present in him. The " light-stop," is a very important early sign if we happen to get it, and it may at once indicate the meaning of irregular pains, or obscure " vago-accessory " disturbances, which otherwise might be unintelligible.

Tabes is usually a very long disease. Some terrible cases run their course shortly, but, as a rule, they run for ten, fifteen, or, not uncommonly, for twenty-five or thirty years. The preliminary stage may be of strangely long duration. The rector of a village in Yorkshire once came to me and said, " It is really absurd my going to any medical man again; I have been to all the medical men I could think of, and I have been to every bath in Europe which I can think of: but I can get no relief from sciatica." After a little while I made out that the sciatica was bilateral. Now I need not tell you that " sciatica " is rarely distinctly bilateral, and that a man with bilateral " sciatica " probably has tabes or other disease of the spinal apparatus. I made out that for eighteen years he had had fulminating pains which at times were intensely severe, making his life miserable. He had not the smallest affection of his gait, but he had the Argyll-Robertson pupil, and the knee-jerks were absent. A year or two afterwards he became unable to wash his face in the morning without falling into the basin. Here, I may say, by the way that some few tabetic patients walk as well or as badly in the gloom or dark as in the light.

How are we to distinguish tabes from neuritis? I must first tell you that you may, and very frequently do, have tabes, neuritis, and general paralysis—all three diseases—mixed up together. Frequently you cannot say " This is not neuritis but is tabes," or you cannot say, " This is general paralysis, and it is not tabes dorsalis "; for you may have any combinations of them. You may have tabes and neuritis together; you may have tabes and general paralysis together, and so on. As you see from the neuritic patient before us, the mere absence of knee-jerk does not prove the presence of tabes, but the " light-stop," so far as I know, is never present in mere neuritis. If the pain in the legs be mild it may be similar in the two diseases; but I think we may say that girdle pains are always absent in peripheral neuritis.

I never heard anyone with this disease complain of girdle pains, but those who have spinal disease generally complain of it, whether you ask for it or not; and by it you may distinguish simple neuritis from neuritis complicated with posterior sclerosis, or from tabes dorsalis pure and simple. Then in neuritis again you may find tenderness in the nerves, and you may test for the reactions of degeneration; but the presence of neuritis would not prove the absence of tabes. I need not say that atrophy of the optic nerves by no means puts mere peripheral neuritis out of the question. In general paralysis you may find other difficulties: you may have the " light-stop," and you may have the " knee-stop," and, as I have said, posterior sclerosis may well co-exist with general paralysis. If, however, you find that there is a little defect of articulation, or any characteristic mental change, then, of course, you have no doubt about general paralysis as one factor in the case.

It is said that if general paralysis be complicated with tabes, and the tabes come first, the disease will be more serious and rapid. This, in my opinion, is by no means always the case. Again, in general paralysis we may distinguish between the sudden falls or drops from which the tabes patients and those of general paralysis suffer. In both diseases patients drop, but the drop in general paralysis is a " pseudo-apoplectic " drop. The patients suddenly turn giddy or more or less unconscious, and come to the ground from the head. They do not hurt themselves, but sink down rather as an apoplectic patient does. A patient with locomotor ataxy, as I have said, generally drops as with a trip, and goes down sharply without any disturbance of brain. The kinds of fall are very easily distinguished in our clinical experience.

To return to our patient, he has rather markedly the " delayed sensations," and especially the perception of a point after a little delay as a rather sharp pain. We estimated this afternoon roughly that two or three seconds would elapse between

the touch of the point of my penknife, and his suddenly drawing himself up and saying it hurt him. The perception of that light touch with the sharp point, after an interval of two or three seconds, is translated into acute pain, and such an illusion of pain from the one touch may persist for no little time. But one other important symptom have I time to mention: You know that what we call the "muscular sense" is often very defective in these cases. When I tell this patient to close his eyes and to put his hands out, he loses sense of where his fingers are, nor can he readily touch his nose when his eyes are shut. You observe his characteristic hesitation. I now show you also that he cannot tell where his legs are when they are placed by us in varied positions, although some of them are positions of awkward constraint. There is an amusing story told of an incident which occurred at the baths of La Malou, whither patients suffering from tabes frequently resort. There is a general bath, in which the patients congregate. One day a leg was seen sticking up in the middle of the group in the bath, and everybody began asking his neighbour whose leg it was. They said, "Here is a leg. Whose is it?" Nobody, however, would own it, and there it remained. To end the matter some impatient attendant seized the leg and pushed it under the water, but its owner never recognized it. This incident illustrates how complete the loss of muscular sense in a tabetic patient can be.

Our time is now up, but even so, sufficient remains to say all I have to say about treatment. Of tabes dorsalis my experience has been large, and I give and have given my patients very honestly the remedies which my betters recommend, for I would never omit any means of relief which might possibly be of service in cases so sad. Under the use of such remedies I have seen the disease after its fashion, run, creep, fly, stop, and behave with all degrees of irregularity, but I have never seen reason to believe that the remedial means used had the smallest influence upon the course of it. One great service, however, can be rendered to the patient, namely, by the use of all means, whether medicinal, mechanical, or moral, to keep all nervo-muscular parts not directly implicated in efficient function. Thus, the disease is kept down to its lowest terms, and the patient's general state up to its best. There is a great disposition in patients of this class to slip into more palsies than are due to organic disease.

SOME RECENT ADVANCES IN THE SURGICAL TREATMENT OF ENLARGEMENT OF THE PROSTATE.

A Paper read before the Eastbourne Medical Society, Oct. 30, 1894, by

C. W. MANSELL-MOULLIN, F.R.C.S.,
Surgeon to the London Hospital.

MR. PRESIDENT,—The subject of treatment of enlargement of the prostate is one that has engaged my close attention for some years past. Through the kindness of many of my friends in the profession I have had what I believe to be exceptional opportunities of observing it; many cases have been placed directly under my care; a large amount of material in the way of notes and records has been placed at my disposal by other surgeons, to whom I am deeply indebted for the trouble that they have taken; and as a result I have arrived at the conviction that, notwithstanding the recent advances in the treatment of this complaint, there may be, and I hope there are, greater improvements still in store for us in the future, and that much more might be done than is done at present towards relieving the distress and prolonging the lives of many of those who suffer from it.

Every one will, I believe, admit at once that the present system of treatment by catheter is not a universal success. It is perfectly true, as Mr. Buckston Browne has pointed out, that there are numerous instances around us of elderly men, active in all the pursuits of ordinary life, and many of them exceptionally well placed in the conduct of affairs, who are obliged to pass all their urine by catheter, and who lead comfortable, useful, and happy lives. But there are many others, and, I believe, a much larger number, of whom, from the very nature of things, much less is heard. The successful ones are living and always in evidence before us, and this alone is apt to lead us unconsciously to over-estimate the proportion that they bear to the rest. Even in them, however, the end, though it may be delayed, is nearly always the same at last. The instrument has to be used more and more frequently as age advances, the difficulty of introducing it becomes greater and greater, the patient loses the power of giving the close personal attention that habitual catheterism requires, and septic cystitis and septic nephritis follow as a natural consequence.

I do not mean to say that the fault is always or entirely that of the catheter: in many instances it is due to delay, to the catheter not being employed sufficiently early, or to carelessness and neglect of the ordinary principles of cleanliness. But after making full allowance for this, it must be admitted

that the introduction of a catheter is always a serious matter, and that its habitual employment is often a remedy scarcely less dangerous than the disease. In enlargement of the prostate the mucous membrane of the bladder is so irritable that even when the instrument is scrupulously clean it is always liable to cause cystitis. The cavity, when there is a post-prostatic pouch, is never emptied as it ought to be : there is always a deposit of mucus left behind. Habitual catheterism destroys the tone of the bladder. According to Sir H. Thompson, the muscular coat may lose its power of contracting absolutely within two years. And undoubtedly habitual catheterism helps in producing the contracted granular kidney that is so often met with in association with enlargement of the prostate. The persistent irritation of the deep part of the urethra maintains a condition of chronic renal congestion which ultimately ends in this.

Now, the primary cause of the cystitis, of the need for a catheter, and of all the consequences that follow from it, is the obstruction caused by the enlargement.

It acts in two ways. In the first place it throws an extra amount of work upon the bladder at a time of life when muscles do not readily become hypertrophied. In the second it keeps the mucous membrane and the muscular coat in a state of chronic congestion, so that their nutrition becomes impaired, and they lose the power of resisting the pyogenic organisms, that sooner or later are sure to gain access to the interior. This is due to the position and arrangement of the prostatic plexus. In childhood and in youth it is only of moderate dimensoins, and according to Hurry Fenwick, all the veins that enter it are accurately valved. In old age the valves disappear, the plexus enlarges to an enormous extent, and lying as it does at the lowest point of the venous system of the pelvis, surrounded by organs of which the blood supply is liable to the greatest variations whenever there is the least straining; and congestion, stasis, and thrombosis follow as they do under the same conditions in the neighbouring bowel. This congestion is, as a matter of fact, the immediate source of nearly all the symptoms assigned to the enlargement. It narrows the exit from the bladder, and makes the obstruction worse. It renders the neck of the bladder irritable, so that the muscular coat is always contracting. It keeps the surface of the mucous membrane in a state of chronic catarrh, so that the epithelium is badly developed, and can no longer protect the deeper structures against micro-organisms. It renders the walls of the bladder hard and rigid, and by impeding the circulation it impairs the nutrition of the muscular fibres to such an extent, that it ends not unfrequently by causing complete atony.

The enlargement that causes this obstruction and congestion is purely and entirely a local growth; it is not dependent on any constitutional cause. Old age has nothing to do with it. Extreme old age, indeed, is nearly exempt from it. I have met with many instances of considerable enlargement in men under fifty years of age, in whom, too, it had already lasted some time, and many others have been recorded by other surgeons. Atheroma has nothing to do with it. That the two diseases frequently occur together is true, but there is not the slightest evidence that the one is the cause of the other. Nor has gout or any other diathesis. Probably it is dependent upon some change that takes place in the testes, but this I shall come to later ; it is sufficient for the present that it is purely local, and that by the way in which it obstructs the exit from the bladder it is the primary cause of the straining and congestion, and of all the consequences that follow from them. A catheter meets this condition very imperfectly. It is true that it empties the bladder more or less completely, but it does not prevent straining and congestion, very often indeed it makes them worse. The question therefore naturally arises whether seeing the immense advances that have been made of recent years in other branches of surgery, it would not be possible, without undue risk to life, to remove the obstruction and relieve the patient once for all from what is almost certain to prove the cause of his death at last, and after great suffering.

Of the many different methods that have been devised with the view of dealing with this obstruction, there are three, and three only, I think, that are in any way likely to meet with general acceptance. Some of the others may be tried now and then, but only under exceptional conditions. These three are a modification of Bottini's ; devised by Watson, of Boston ; McGill's operation, or suprapubic prostatectomy and removal of the testes.

Bottini makes use of the galvano cautery through the urethra. One of his instruments is shaped like a sound with two small platinum plates fitted into its concavity. They lie parallel to each other, about one m.m. apart. Each is about $\frac{1}{8}$ c.m. in breadth, and two in length. The ends that lie near the beak are joined together : the others are attached to two copper wires running in the shaft which also contains a pair of tubes for the circulation of cold water. In the other instrument the cautery, which is bent round into the form of a loop, is concealed and insulated in what would correspond to the male blade of a lithotrite.

The current required must be of considerable strength, sixteen to twenty ampères. Bottini makes use of accumulators, but if the patient's house is supplied with the electric light, and the current is an alternating one, the required strength can be obtained by connecting it to a Woake's transformer.

No anæsthetic is required, and no water need be injected into the bladder, but it is as well that the urethra should have been accustomed to manipulation. When the point is in the bladder, it is turned downwards as in searching for a stone, and gently drawn outwards until it is felt that the beak is in contact with the obstruction. If there is any doubt, the exact position can be ascertained by means of the finger in the rectum. The current is then switched on, and the curve pressed against the obstructing body, a stream of water being made to circulate through the instrument to prevent the shaft becoming too hot. By gently elevating the handle, the platinum can be made to burn the required depth. When this is reached, the instrument is pushed into the bladder and allowed to cool. If the scab sticks to it, it must be burnt off.

Bottini claims that he can burn a track two centimetres in depth in two minutes. The scab usually separates about the tenth or fifteenth day. There is sometimes a good deal of strangury after the operation, and secondary hæmorrhage has occurred.

Bottini has operated upon seventy-seven cases, of these two (these were among the earliest) died. In twelve there was no result; in eleven some improvement, and in fifty-two complete cure. The results obtained by other surgeons vary considerably and are not so good on the whole.

The reason for this is not far to seek. Bottini's operation is of no use in cases of advanced disease. For these, Bottini recommends tunnelling. Nor is it of any use when the growth is mainly vesical. A small obstruction confined to the posterior wall of the urethra at the orifice of the bladder can be dealt with effectually, but such cases are rare, and difficult to diagnose.

On the other hand, in the earlier stages of the disease, when the congestive symptoms and not the purely obstructive ones are the most prominent, it may be of great service. The cautery cutting into the mucous and submucous layers destroys the venous plexuses, and the dense cicatrix that it leaves completes the work. It does not stop the growth of the enlargement, and can have but little effect upon valvular projections, but it does prevent many of the distressing symptoms to which the enlargement gives rise, and by stopping the congestion it can at least delay the consequences that follow.

This end, however, can be accomplished very much better, and with much greater certainty and safety, by adopting the plan recommended by Watson. The urethra is opened in the perineum, and the cautery introduced through the incision. The wound is exceedingly small and heals without any difficulty, but it enables the finger to be introduced, so that the condition of the neck of the bladder and the prostate can be ascertained with accuracy; it does away with the objection that the operation is performed in the dark; the bladder can be drained if necessary, and any hæmorrhage that occurs either during the operation or later, when the sloughs are beginning to separate, can be dealt with more easily.

When the enlargement is confined to the lateral lobes this method is not of much service; but in some cases a certain amount of benefit has been derived from the division of the fold of mucous membrane that is raised up between the projecting ends of the two lobes behind the orifice.

Whether the relief obtained by this operation is likely to be permanent in all cases, time alone can tell. Probably it will not be, for although the congestion is checked, the growth of the gland is not. But at least it is of some advantage, with so little risk, to be able to dispense with the use of a catheter for some years, and to be in no worse a position at the end of that time.

McGill's operation has a much higher ideal, it aims at removing the whole of the obstruction, and restoring the outlet to its original shape, if not to its original flexibility. It is performed through a suprapubic incision. Provided the respiration is sufficiently good, the patient is placed in Trendelenburg's position; an opening is made in the bladder, and the sides of the incision are fastened by temporary sutures on either side to the skin. There is no need to use a rectal bag. On several occasions on which it has been employed it has proved itself a source of danger, and if the bladder is of reasonable size, it can always be dispensed with. If the bladder is not of reasonable size the operation is impracticable; it is impossible to carry out the necessary manipulation unless it will hold from eight to ten ounces at the least.

The growth must be removed as far as possible by enucleation, after the mucous membrane covering it has been divided with stout scissors. Hæmorrhage in that case is rarely severe, for the prostate itself, unless it is inflamed or congested, is not a very vascular organ. How much should be removed varies with each case. In one McGill removed a fragment only the size of a pea; in another he took out four ounces; but if the operation is to be permanently successful the whole of the vesical outgrowth must be taken away, the upper part of the projecting lateral lobes enucleated (this can usually be done without much difficulty through the median incision), and if the posterior wall of the urethra is hard and rigid, this must either be incised or a deep groove must be cut in it with the actual cautery. In many instances it is advisable to make a perineal incision for the sake of drainage, and this can then be utilised to ascertain if the urethra is perfectly clear. The structures around the neck of the bladder completely lose their elasticity, and unless the opening is made sufficiently funnel-shaped, relapse is very likely to occur.

This operation is, of course, a very much more serious one than that described already, but it is very much more thorough. Lateral as well as median lobes can be dealt with equally well. The "perineal distance," which is such a bar to perineal prostatectomy, is of no consequence, except in so far that it indicates a great increase in the bulk of the gland. The interior of the bladder and the prostatic urethra can be examined; calculi, which are much more common in cases of prostatic overgrowth than is usually believed, can be removed; phosphatic *débris* scraped from the walls; the exact amount and shape of the enlargement, and the relation that it bears to the cavity made out, and the effect that the removal of the obstruction has upon the outlet accurately gauged at the time, so that no more and no less than is necessary is done. Vesical outgrowths can be dealt with in no other way.

The rate of mortality, it is true, has hitherto been very high; but this has arisen from the fact that the operation has often been put off until the patient was actually *in extremis*. Of the ninety-four cases that I collected for my Hunterian Lectures, 20 per cent. died; but the mortality in the first half was 25 per cent., in the second half 15, and since then it has fallen much lower; Mayo Robson, for example, has operated eleven times with only one death. Moreover, there are two recent modifications which will, I believe, prove of very material service. One consists in performing the operation in two stages—waiting after the bladder has been opened until the patient has regained his strength to some extent, and the urine has lost its septic character; the other, which has been tried on several occasions by Nicoll and Macewen, leaves the mucous membrane of the bladder intact, and so preserves the wound from all contamination. The bladder is opened supra pubes, and the fingers of the left hand introduced into it to act as a guide; but the actual removal of the prostate, through an opening in the perineum, is effected by cutting forceps, shaped something like a parrot's beak. In this way the whole obstructing mass can be taken away, and the bladder kept intact.

The cases that are most suitable for suprapubic prostatectomy are those in which there is a valvular upgrowth, or three upgrowths meeting together over the orifice when the bladder contracts; those in which there is great thickening of the median posterior wall of the urethra, shown by the way in which a catheter is obstructed; and those in which there is an intra-vesical outgrowth, causing intense irritation. Cases in which there is much difficulty in passing a catheter; in which there is frequent hæmorrhage, or where the patient will not take the necessary precautions; and those in which the muscular coat of the bladder is beginning to fail, or the specific gravity of the urine is beginning to fall, should also be advised to undergo operation but though my own preference is for the suprapubic route, the perineal one may answer for these, provided the growth is small. Persistence in catheterism in such cases can only lead to disaster. When septic cystitis has already broken out many times, or has lasted more or less continuously for many years; when the bladder is hopelessly ruined and coated with phosphatic *débris;* when, in short, the case has reached that stage which has hitherto been thought to justify the performance of the suprapubic operation, a totally different line of treatment should be recommended.

This brings me to the third of the three great improvements in the treatment of prostatic enlargement that I mentioned at the beginning. It has now been proved conclusively that removal of the testes is followed by complete disappearance of the enlargement. It is not the shrinkage that has been noticed in a good many instances after partial operations, such as puncture or drainage; it is atrophy of the most complete character, and the most rapid progress. Within three weeks a growth the size of a Tangerine orange has disappeared altogether, and even the day after the operation there may be a perceptible difference. The idea of the possibility of such an occurrence appears, as frequently happens, to have sprung up in the minds of several people at the same time. I discussed it with a patient in November, 1892, shortly after the publication of my Hunterian Lectures, but as I had no solid groundwork of facts for my argument I am not surprised the patient was not convinced. Ramm, of Christiania, however, performed the operation twice in April, 1893, with complete success; and later, in June, 1893, Professor White delivered an address before the American Surgical Association, in which he detailed a number of experiments that he had performed with this view, without, however, having been led to any definite conviction upon the subject himself. Since then it has been performed five times in America, and twice in England by myself. One of my patients was eighty-one years of age, the other seventy-four. The former had suffered from repeated attacks of retention; catheterism was almost impossible, owing to the shape and size of the growth, and suprapubic puncture had already been performed by one of my colleagues. He recovered perfectly. The latter had suffered the most fearful agony for many years from cystitis and strangury, and had been compelled to wear a portable urinal strapped on to his legs. The operation in his case was only performed four days ago, but already there is distinct amelioration of the symptoms.

Whether this operation will succeed in all cases alike it is not possible to say; at least it has succeeded in every one in which it has been tried.

It is true that the bladder has not recovered perfectly in all, but this is not surprising. After years of intense septic cystitis the muscular coat is practically destroyed, and removal of the obstructing body and cessation of the cystitis of course cannot restore it. But even in those cases the relief gained is most valuable for catheterism, when the prostate is atrophied, and the congestion of the neck of the bladder has disappeared, is a very different matter from the same thing when there is enormous and obstructive enlargement.

The operation is not one to be recommended except under special circumstances, when everything else has been tried and failed; and in all cases the final decision must be left to the patients themselves. But when the alternative is certain death at no distant period, by what has been aptly called a slow but fatal form of torture, I believe that we are amply justified, not only in laying the choice before the patient, but in strongly advising the operation.

Unhappily, for there is reason to believe that many patients would consent to this at an earlier stage in the disease, division of the vasa deferentia does not appear to be of the same value. It has been tried, but though it appears to have succeeded in some cases, it certainly has failed in others; and it is noteworthy that according to some authorities, division of the vas deferens is not invariably followed by atrophy of the testis. It seems probable that the influence exerted on the prostate by the testes makes itself felt through the medium of the nervous system. Unilateral castration, according to Watson and Launois, is followed by unilateral atrophy. There may therefore be a future for ligature of the whole spermatic cord, not the vas deferens only; a plan that was suggested to me by Mr. Howlett, of Hull, and which seems worthy of trial in cases in which consent cannot be gained for the more complete operation of removal of the testes.

A CLINICAL LECTURE

ON

DISEASES OF THE SKIN DUE TO PUS-COCCI.

Delivered at University College Hospital on Saturday, November 17th, 1894, by

H. RADCLIFFE CROCKER, M.D., F.R.C.P.,

Physician to the Skin Department in the Hospital.

GENTLEMEN,—The study of bacteriology has produced changes in our knowledge of disease in almost all forms and regions of the body, but in no class of diseases has it produced a greater revolution than in diseases of the skin, both as regards our understanding of the morbid processes, and also improving our treatment. This is very natural, as the skin is so constantly exposed to parasitic infection. Whenever there is a disturbance of the surface, either from inflammation or even from mechanical injuries, it is at once exposed to an invasion of various organisms which are almost ever present in the atmosphere. Of all the diseases thus produced, those due to pus-cocci are perhaps the most definite, and those affording most satisfaction with regard to treatment. There are several diseases which we now know to be due to pus-cocci, and by these we chiefly mean staphylococcus aureus et albus—organisms which are probably, although different in colour, of exactly the same species, only perhaps modified by environment. Of the diseases of which I am about to speak, some we know to be due to these organisms, and in others the inference is very strong that they are also due to it. In addition to those which are directly due to these staphylococci, there are others which are secondarily invaded by them, in which, therefore, considerable modifications are produced. Of course, many of you are aware that pus may exist quite independently of pus-cocci, that irritants of chemical and even mechanical origin will produce suppuration, and that we get it from other organisms, for instance, in connection with tubercle. So you must remember that there are pustular diseases in which there are no pus-cocci present, at all events at first. But the diseases which we specially allude to are those comprised under impetigo contagiosa—pustular eczema, boils, carbuncles, and fourthly, certain cases of folliculitis, especially those of the beard, which were included formerly under the term of non-parasitic sycosis, as distinguished from that form of parasite sycosis, which was up to the present time the only one known to have a parasite. The latter form of sycosis was hyphomycetic, i.e., due to a fungus—viz., that of tinea tonsurans. But the other forms we now know to be equally parasitic, since they are due to pus-cocci or schizomycetic. Hitherto we have only called skin diseases parasitic which were due either to animal parasites or to those traceable to fungi. That was partly, of course, because our knowledge was not sufficiently definite to make other classes, and partly because no doubt three-fourths of the inflammatory diseases have more or less to do with microbes, though like the one of pityriasis rosea you have seen this morning, we do not yet know what particular microbe produces it. To return to the subject, the main difference between impetigo contagiosa, boils, and carbuncles, is due to the anatomical seat of the lesions and the mode of entrance of the organisms. In impetigo contagiosa only the most superficial layers are

affected, and the microbes generally gain entrance either from antecedent inflammation or some mechanical abrasion, or direct inoculation by the nails, which may have been previously contaminated by pus from other sores, or from microbes which are always present in the air and generally present under our nails. You know that surgeons nowadays are specially careful to clean their nails immediately before performing operations.

The lesions of impetigo contagiosa are extremely variable. As most of you know, I have great difficulty in getting you to always distinguish it, even if you have half a dozen cases in a morning ; perhaps two of the half dozen will be missed, even by men who have seen the other form, on account of its extreme variability in aspect. Some of the varieties you may see here (cases and drawings shown). The case of this child is what we may call typical, so far as it affects the head. In all cases are separate lesions quite superficial in the skin, and which, though they commence as vesicles, or perhaps pustules, dry up into scabs with little or no inflammation about them. Lesions produced by accidental inoculation by nails or other means, have, of course, no special distribution, and it is this character of being scattered about the surface that makes an important point in their diagnosis. The next thing is, when they occur about the face, there is generally very little inflammation round the lesion, especially at first, and so the scabs simply appear as if they were stuck on ; they have the same appearance if removed from one part of the face and put on another. When they have been present a little while, they may get red and slightly inflamed. Here we have cases where, although there has been a very large number of lesions, there is very little redness and inflammation present ; in this child there is scarcely any redness in the lesions. We do not always get it in the vesicular stage when the child is brought to us ; the vesicles have often been ruptured before they come, because some little irritation has occurred, and the spots have been rubbed. These vesicles may be small or large, and the larger forms often get mistaken for varieties of pemphigus, and cases reported as pemphigus contagiosa are frequently bullous forms of impetigo contagiosa. Here are two examples. The commoner form I have not a drawing of, but we see them almost every day. When impetigo contagiosa occurs on the limbs it always gets rubbed by the clothes, and you get this condition, which is often called ecthyma : but really my belief is—and I am satisfied it is true—that ecthyma is simply impetigo contagiosa which has been rubbed into a condition of additional inflammation. Some authors still hold that there is an ecthyma separate from impetigo contagiosa, and that there is always a pustular margin which tends to spread peripherally. But that is true of impetigo

contagiosa, as I will show you presently, and they are so frequently associated together—these lesions on the face and on the body—that I cannot understand how any one should doubt their being really identical diseases from a clinical point of view. They also both call for the same treatment.

Here is an instance of a rare variety of impetigo contagiosa, and one which might easily give rise to difficulty—in fact, did so. This commenced as flaccid bullæ, which dried and formed lesions such as we see there—a scab with a vesicular or a pustular margin as the case may be, and that surrounded by a red areola. That eventually cleared in the centre, and then you had a comparatively smooth centre, a crusted irregular collar surrounding it, and again a fluid border and the red areola. Eventually the disease spread all over the back in a form much more complicated than we have in the drawings. That this was due to the staphylococcus aureus, was proved by inoculating fluid from an unruptured bullæ and cultivating it ; and practically a pure culture of staphylococcus aureus was produced. There is a tropical variety, evidently of this kind, which was called by Dr. Manson pemphigus contagiosus tropicus ; we may go into that another time. These again are examples of so-called ecthyma in the head. In one you see the pus-cocci again enters, without any abrasion of the skin ; and we see, therefore, it may occur in connection with vaccination. Here is an instance of vaccination lesions ; pus-cocci got into them afterwards, and then produced the pustules which dried up into these greenish scabs. The mother would say " It is due to vaccination with bad matter," but really it is due to the arm not being protected from the cocci in the atmosphere, and if it were always carefully covered with alembroth wool, such lesions could always be avoided. The fact that you always have to bear in mind is, that it does not matter how a breach of surface is produced, if pus-cocci happens to get into it, it will produce the lesions which we know as impetigo contagiosa or ecthyma. You never get any scarring from this sort of lesions, unless some of them are violently scratched, and damaged in that way ; but from the lesions themselves you never get any scarring, only a slight stain. But in boils, where the process is somewhat deeper, you do get, of course, marked scarring ; for while in boils the same parasitic invasion occurs, it appears to travel along the follicles, and so you get it deeper in the tissues. In carbuncles there is the same condition, due to the same cocci, only there the lesions are in the subcutaneous tissue, while in boils they are in the cutis vera generally, although they may spread to the subcutaneous tissue. Carbuncles only occur where the skin is thick, and the cocci travel deeper down. It is said by an American author that it travels along fat columns, which go from the hair follicles into the sub-

cutaneous tissue; then in the subcutaneous tissue it excites the conditions which we know clinically as carbuncle. Then we come to these forms of folliculitis specially affecting the hairy parts of the face. No doubt there are others elsewhere also, but these are a type of them. Here, of course, it gets into the follicle, and gradually spreads from follicle to follicle until it affects the whole of the hairy parts of the face; they are all of the same kind. They lead to scarring and, of course, complete destruction of the hairy follicles in which the suppuration occurs, and they differ from hypho-genic sycosis in this point—the hyphogenic sycosis is a much more acute process, and generally occurs in multiple foci.

Moreover, the hyphogenic sycosis sets up a more active form, and you get large pustules or groups of pustules, beginning, of course, in one spot, but speedily inoculating itself into several others and producing much more loosening of the hair than you get in the ordinary sycosis. One of the tests that used to be applied was that in the hyphogenic form the hairs could be pulled out almost painlessly with the forceps, whilst in what was called the true sycosis this could not be done. That, however, is only relatively correct. It is quite true that it is pretty painful to epilate in coc-cogenic sycosis, and that with the other you can pull the hairs out more readily; but there are ex-ceptions both ways, and if the inflammation is not very great in the hyphogenic form, of course, it is equally painful. Still, if a large number of the hairs come out easily without pain you may be pretty sure that is due to the fungus and not to the coccus. Of course, the microscope will decide in these cases, but it is not always quite as easy as it seems, for in many cases, if the hairs pull out very easily without any sheaths you may find no fungus adhering to them, and the fungus does not in all cases penetrate into the sheath itself. In the coc-cogenic form the cocci are always in the sheaths, which generally adhere to the hair when it is pulled out. Now, as a secondary invasion, we see them occurring in a great number of cases; perhaps eczema is the most typical. All forms of pustular eczema are instances of secondary in-vasion; first of all, there is the ordinary simple inflammation, of which this is an example—then invasion of pus-cocci, producing this thick crust of which all these drawings are examples. These cases were formerly called impetigo facei; this was called eczema impetiginodes. Now, to avoid any confusion of terms it is better to speak of them as pustular eczema, and to avoid the use of the word "impetigo" pure and simple, because if you say "impetigo" people are not quite sure whether you mean the old pustular eczema, or what we now call "impetigo contagiosa." I, therefore, always insist that you should use the term "impetigo contagiosa," because impetigo

was applied to almost anything pustular in former days.

You see then that this generalization is an important one, and is reflected in the treatment. The principle of treatment in all these cases is to destroy the pus-cocci, which are the origin of the disease, and the difference lies only in what is the most convenient method of doing it in the different cases you have to deal with. In these superficial forms all you have to do is first to remove the crusts; this may be done in cases like the one before us, by soaking in carbolised oil. If it is on the face or surface generally, simply bathe with warm water until the lesions can be picked off. On the scalp the best way is to dip strips of flannel in carbolised oil—1 in 60—and soak them for several hours, and then with a pair of scissors cut the hair beneath the crust and remove it. You must always insist upon the removal of the crusts. If you simply tell the mother to take off the crusts, she either bathes them for a little while or puts the oil on for a little while, and because they do not drop off she says they won't come off. You must explain to her exactly how it is to be done. And when you have removed them, the remedy I always use, because it is convenient and it is absolutely infallible if properly applied, is a diluted am-moniated mercury ointment. It is clean and is absolutely effectual if the parts are constantly soaked with it, so that they are never allowed to dry again into scabs. Where we have a crusting on a large surface then it might be treated by sponging with perchloride of mercury solution—1 in 2,000 would be about the right strength.

I treat all these cases of ecthyma and impetigo contagiosa with the diluted ammoniated mercury ointment—15 gr. to 1 oz. of lard. In boils and carbuncles the method is different. In former days efforts were always made to purify the blood, as the cause was said to be impurities in the blood. So far, they were right, no doubt; but the im-purities were the staphylococci, and we can exhaust the supply of staphylococci by continuous and systematic local disinfection. The best way to treat furunculi is simply by external means, and not as they did in former days, which was uncertain and tedious; the plan adopted to-day is to open every boil as soon as it appears, syringe it out with some carbolic lotion—1 in 60—or rub in iodoform. After this is done, the bullæ soon cease to appear, and the supply of cocci will be exhausted, for each boil is a breeding ground for a fresh supply of staphylococci. Above all, never poultice a boil with linseed meal or bread, but if there is great pain you may apply an antiseptic one, such as a warm boracic acid fomentation. But even then, the best plan is to open each boil and clear it out as soon as possible. It is surprising how soon the disease may be cured if this is done systemati-

cally. In ordinary boils they generally come out one or two at a time, and there is no difficulty in doing so. There is another disease allied to the ordinary boils—very probably due to the same cause, though of that we are not certain—and these are the condition called sweat boils or hydrosadenitis. These come chiefly in the axillæ, groins, and flexures, where the sweat glands are large and abundant. They form red swellings, but are not very painful, and have not the hardness of an ordinary boil. They rupture and evacuate their contents spontaneously in most instances, and that particular one gets well, but the process may continue for a considerable time. I have known a case in which all the flexures were riddled with holes and sinuses from this process having been allowed to go on for a great many years. It was that of an elderly lady who I saw with Dr. Duncan Greig. She had had medicines of all kinds and everything that the ingenuity of many London physicians could suggest, but without any benefit. But when I had all these boils systematically opened, the sinuses split up and disinfected with iodoform and carbolic lotion, in about three months the disease was perfectly cured. Although the staphylococcus aureus has not been proved to be the cause of these cases, it is extremely probable that it is so, and at all events in this particular patient there was a secondary invasion of pus cocci, even if not a primary one.

In carbuncles, again, we have to modify the treatment, though the principle remains the same. In a spreading carbuncle, the best way to stop it is by injections of carbolic acid—a pretty strong one is desirable—1 in 30. Two or three minims injected at short intervals round the carbuncle will speedily prevent it spreading. In the back they may become of enormous size as big as a dinner-plate sometimes, and endanger the life of the patient.

Not so very long ago, we had a man here with a very large one; he would not come into the hospital at first, and it spread from about the size of an orange to the size of my hand. He then came in, and we injected him with carbolic acid in this way. It was so large that we did not like to go all round at once for fear of symptoms of poisoning, but the local effect of the carbolic acid made the demonstration all the more striking. Where the carbolic acid was injected, it stopped it at once, while the side that was not injected continued to spread. By the time we had formed a cordon of injections right round, all the activity of the process stopped. There was then only a huge slough, which we could cut away freely, leaving a deep irregular crater; but this speedily shrank, and the swelling round subsided, leaving only a simple wound to deal with. In addition to injecting, the skin round the sinuses were filled with iodoform—the best disinfectant for the central portion of carbuncles. No doubt in many boils and carbuncles,

there may be some pre-disposing influence which makes them flourish more freely, such as diabetes mellitus, and not only in cases of boils and carbuncles, but in many other diseases too you should examine the urine. But far more frequent than diabetes is sewer-gas poisoning, and it is therefore quite as necessary to examine the surroundings of your patient as to examine your patient himself. Of course, all such causes should be removed.

Now then, again with regard to eczema. The pustular eczema, which was generally thought the most formidable, is now the least difficult of treatment. In all these crusted cases the best way is to remove the crusts by soaking with carbolized oil, or, if you like, a soft antiseptic ointment ; soften the crusts, pick them off, and then use the iodoform ointment—5 gr. of iodoform to 1 oz. of lard. Spread it on strips of lint, and lay it evenly all over the head of a child for instance, with pustular eczema. In the course of a week, if it is thoroughly applied, the pustular element will be practically destroyed, and the condition will be reduced either to a simple inflammation, which will then get readily well with any one of the numerous astringent and protecting ointments, like zinc, lead, or boric acid ointment, without any trouble, while in many cases the inflammation will have dried up altogether. Of course, in some cases simply the pustular element is removed, and you have still to deal with the ordinary eczema, and whether it is connected with internal causes will be a matter for you to investigate. But in any case always, first of all, remove all the pustular element, and bring it down to that condition, and, as a rule, it is quite dry by that time, although some little inflammation will remain.

With regard to these cases of sycosis, the treatment is again on the same principle, but epilation plays an important part. The patient should be instructed to carefully epilate a definite area every day—take one square inch and clear that thoroughly. It is no good to pull out a few here and a few there ; but a patch must be cleared away thoroughly, and then you can apply one of the numerous parasiticides—iodoform is objectionable, I confess, but it is useful enough—or you try some of the numerous substitutes for it, though there are none quite equal to iodoform. One of the newer ones is loretin, used in about the same strength ; it appears to be effectual and unobjectionable. Or, again, you may use a weak carbolic acid and sulphur ointment — about 5 gr. sulphur and 10 min. carbolic acid to 1 oz. lard, rubbed in after epilation ; or one of the numerous mercurial ointments—1 per cent. oleate of mercury or 2 dr. nitrate of mercury to 6 oz. of lard. Any one of these is effectual if care is taken that it actually does penetrate and get into the site of the disease. Therefore, it should always be rubbed in directly after epilation, as well as at other times

when epilation has not been practised. You should impress upon your patient the necessity for thorough and complete epilation, and keeping it epilated for a considerable time, and cases that have lasted for years may then be thoroughly eradicated; but it is always troublesome, because you are never quite sure you have destroyed the last coccus, owing to the difficulty of getting down to it; but still, steady perseverance and systematic treatment will cure even the most obstinate cases.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Case illustrating the Diagnosis of Scirrhus of the Breast in an early stage.

The difficulties in the recognition of cancer of the breast in its earliest stages are well illustrated in the following case. I had attended Miss S.'s mother twenty years ago on her death-bed for an abdominal tumour, and it was known that one of her aunts had died from cancer of the breast. Miss S. herself, aged 46, and in good general health, came to me in September last, in great anxiety about her own breasts. She was rather stout, and the breasts were full. In both breasts there were ill-defined indurations. After careful examination, I satisfied myself that in the left, deeply placed, there was a disc of ill-circumscribed thickening, which was probably cancer. Having regard to the patient's family history, I advised a precautionary operation, and to this she readily assented. On the operation table, two or three weeks after my examination of the case, I had forgotten in which breast it was, and the conditions in the right seemed more suspicious than those in the left. The nurse, however, assured me that it was the left breast that the patient expected to have removed, and, on more careful examination, I again recognized the induration which I had formerly discovered. One of my assistants expressed the confident opinion that there was no cancerous growth in either breast. The size of the breast was such that I felt sure I should gain no conclusive information from an incision into it, and, feeling tolerably certain in my diagnosis, I proceeded with the operation. It should be said that there was not the slightest retraction of the nipple. On incising the breast after its removal I found a great number of small cysts containing greenish fluid, and varying in size from a horse bean to a small cherry. The lobules of the breast

were dense, and in some parts almost hard, but their section was white and fibrous, and showed no trace of cancer. For a few moments I thought that I had made an error, and that there really was no malignant growth; but, having turned the breast over and carefully identified the part which, before the operation I had thought most suspicious, I there found a disc of induration, about the size of a half-crown and twice its thickness, a section of which presented, as all admitted, the most characteristic conditions of scirrhus. We have, therefore, a good instance of cancer occurring in association with other non-malignant changes in the gland, the presence of which made its recognition very difficult. As regards the other breast, I have no doubt that there are in it many small cysts, and that, with the exception possibly of the absence of actual scirrhus, it is in the same condition as the one excised. It is now four months since the operation was performed, and Miss S. is in such a condition of continual anxiety about the remaining breast that it will, I think, be the wisest and kindest measure to remove it also. It is, of course, quite impossible, after our experience of the other, to assure her that there is no risk of cancer, or to give any other reply than an affirmative one to her question, "Would it not be safest to remove it?"*

Herpetiform Morphœa affecting the region of the Clavicle.

A woman, aged 67, who had always enjoyed good health, attended in order to show a patch of herpetiform morphœa over her left clavicle. It was in all respects exactly like one of the portraits in the museum (which was shown). There was a large group of white lardaceous spots arranged somewhat in streaks on the upper part of the sternum, and a little crossing the middle line. External to this, passing towards the shoulder, were some smaller groups also arranged in streaks. They were all of ivory whiteness; distinctly perceptible to the finger as polished patches a little roughened, whilst here and there were a few minute pinhole dots. Above the clavicle on the shoulder, there were two groups of congested and slightly pigmented spots without any ivory appearance. Mrs. C.'s father had had shingles on his chest in early life, and when elderly had had a very bad attack of herpes on the forehead. A sister also had had shingles on the chest.

Comments.—No case could better than this illustrate the appropriateness of the term "herpetiform" as applied to morphœa. The groups of little ivory-white spots are arranged exactly like those of herpes zoster, and are wholly confined to one side.

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* Since this was written the second breast has been excised, and found to contain both cysts and scirrhus.

Herpetiform Morphœa affecting the Ear and the Skin behind it.

The subject of this case was a florid little girl, aged 7, in excellent health, brought by Dr. Alfred Eddowes. The skin over her left mastoid process and downwards to the angle of the jaw, was in a condition of white ivory-like smoothness. The external ear, especially at its external border, was shrivelled. No patches had been discovered elsewhere.

By the side of this patient was placed a drawing showing a morphœa-patch located in precisely the same region. In it there was, however, another patch on the opposite side, and passing obliquely downwards towards the shoulder.

Comments.—This case, like the preceding, illustrates the location of morphœa by nerve distribution and its entirely unilateral development. The patch will, in all probability, disappear in the course of years, and no others will follow. It is to be feared, however, that the ear will never recover its plump condition.

Lupus Vulgaris in association with Strumous Disease of Glands.

A fair complexioned woman, aged 32, from Durham, illustrated the coincidence of lupus of the skin with severe glandular struma. Her neck on both sides was very extensively scarred by gland abscesses. These cicatrices were of their usual character and perfectly sound. Near to them however, and on the front of the neck were some very large patches of lupus vulgaris in the exfoliative stage. The patient dated her gland disease from the age of 11, and her lupus from that of 18. Both her parents and seven brothers and sisters were living, and reputed healthy. She herself had never been regarded as consumptive, and the only history of tuberculosis in the family was that a paternal uncle had died of phthisis.

Comments.—This case illustrates one of the remarkable facts in the history of tubercular affections: that the disease of one tissue or organ does not as a rule produce material capable of infecting other and different structures. Glandular struma keeps to the glands, and lupus keeps to the skin. Although we believe that the two affections are very closely allied in their nature, yet we know well that when the skin is affected over a glandular abscess it seldom or never assumes the characters of lupus. In the case before us, the two affections have run their course together, but without mixing. The strumous glands have suppurated and ulcerated extensively, but their scars are wholly different from those of lupus. The lupus patches, although near to, are not continuous with the gland-scars, and, if we may trust the patient's history, the lupus did not begin until seven years after the gland

struma. My theory of strumous and tubercular affections is that they do not depend as a rule upon the introduction of the bacillus from without. Those organisms probably exist in a latent form in almost all persons, and only manifest activity when the general strength is sufficiently lowered, or the vital integrity of some part injured. They then enter into a sort of partnership growth with the cell structures of the part, and the result is inflammation and parasitic development at the same time. Thus the morbid process is infective only to the tissue in which it had originated; and the parasite is not free to wander through the whole organism I have demonstrated here, over and over again, how a patient with a single patch of lupus may have that patch slowly spreading for many years without the slightest evidence of infection of distant parts. It is by reference to the hypothesis, which I have just suggested, that I should seek to explain the differences, which we recognize in the various forms of struma, and particularly in those which the skin offers so abundantly for our study.

Tumour in the End of a Finger.

A stout, well-grown girl of 16 presented a lumpy condition of the end of the left forefinger. The skin itself was not diseased, but in its deeper layers, or in the subcutaneous cellular tissue there was a condition of ill-defined swelling, which considerably deformed the finger end. The pulp of the finger was chiefly involved, but recently the disease was progressing towards the back. The patient pointed to some little dark specks not so large as pins' heads, which, she said, were the first stage of the disease. There were very few of them and they were very ill-marked. The finger was not in the least sore or tender, but the patient had sometimes complained that it was a little numb. The enlargements, which had been present two years or more, were distinctly aggressive and still spreading.

Comments.—I dare not venture in this case any very definite diagnosis; but I have seen cases somewhat similar, in which there was reason to think that the thickening was due to changes of a nævoid or erectile character.

Cystitis of Pregnancy. — Camphor gr. 1¾, opium gr.⅓. To make one pill. Give 5 or 6 daily. If cystitis is purulent, use boric acid injections, 2 to 100.—(*Revue Inter. de Bibl. Méd.*)

Vomiting in Cholera Infantum.—
  Emulsion of poppy seed ... ℥iij½
  Hydrochlorate of cocaine ... gr.¹⁄₁₂–⅙
One teaspoonful every hour.
     (*Therapeutische Monatshefte.*)

THE CLINICAL JOURNAL.

POST-GRADUATE LECTURE

ON THE

TREATMENT OF SOME SPECIFIC FEVERS.

Delivered at the West London Hospital, Nov., 1894,

By SEYMOUR TAYLOR, M.D., M.R.C.P.,

Senior Assistant Physician to the Hospital.

GENTLEMEN,——In considering the subject for my discussion to you to-day I was somewhat at a loss to know what would most interest you ; then I followed my usual plan in similar difficulties, and asked myself, " What subject should I select were I the listener and in general practice ?" That same day I met a friend in consultation over a case of diphtheria in which the diagnosis was evident, but in which the treatment could not be so easily carried out as one would suppose, and he expressed a wish to know what we should do ourselves in ordinary cases of fever. I will, therefore, with your permission, discuss some of the points in the treatment of scarlet fever, diphtheria, typhoid, and whooping-cough.

As regards Scarlet Fever, its prevalence, its infectiousness, and its death-rate have raised it to an importance beyond that of any specific fever. Other fevers are individually more fatal, but not so prevalent. For this reason the mortality bill in scarlet fever is, in the aggregate, the heaviest one of all zymotic diseases.

Firstly, the room must be as large as possible. If you can, let its dimensions approach the 2,000 cubic feet required for the " offensive sick." Indeed, did we look on all specific fevers as coming under the category of offensive sick, and secure cubic space accordingly, our death-rate would be considerably lower than it is. Constant ventilation can be secured by an open fireplace and window. When practising among poor and congested districts in the North I used to take out a window-pane ; this secured a certain amount of ventilation in the sick-room. In scarlet fever there is more danger from an offensive atmo-sphere than from a cold one. This axiom probably holds true in most zymotic diseases ; certainly it is essential in the treatment of small-pox. As regards duration of the fever, although the febrile process ceases abruptly at about the sixth day, the child must be kept in bed three weeks, at least, and must not be allowed to leave the room for six weeks. Indeed, although this period is commonly stated to be the limit for complete desquamation, I find it is not by any means invariably so. The thick cuticle over the heels and on the palms is, with difficulty, got rid of under seven weeks, and so long as a flake remains the patient is eminently infectious.

There is a practice, which is widely adopted, of applying carbolized oil and unguents to the desquamating body. It has never commended itself to my mind. To me it is putting a risk on the patient in order to stay possible infection to others. By such means we check the important transpiration through the pores of the skin which are choked with oil, it may be antiseptic, and hinder an eliminative process in order to gain an advantage which is problematical only. I think it more scientific to bathe the patient in a warm diluted solution of perchloride of mercury (1 in 1000), which is quite as antiseptic as, or more so than, carbolic acid, and the skin's action is not impeded. Another excellent and, indeed, more speedy way of hastening desquamation—though, of course, not antiseptic—is to give a patient, twice or thrice a week, a hot bath, in which a good handful of ordinary washing soda has been dissolved. Afterwards the child is wrapped in a hot blanket, in which it remains all night. On the morrow the blanket will be full of detached flakes, and will, of course, require careful disinfection. As regards drugs, no remedy is required in an ordinary mild case beyond a saline draught every three hours during the course of the fever. One symptom, however, often requires alleviation, and our text-books fail to draw much attention to it. I refer to itching of the skin. This is often almost intolerable, and will keep a child restless and irritable. I find nothing more useful than sponging the body with a warm solution of carbonate of soda (gr.x to the oz.), to which a little mucilage has been added. It forms a soothing and grateful application to an inflamed skin.

As regards the complications of scarlet fever, rheumatism is more frequent than is generally supposed; there is some relation, at present not made clear, between the throat and the joints, and scarlet fever is one of those diseases in which this relation is manifest. I find salicin and the salicylates most useful; bromide óf ammonia may be added to procure sleep. In cases in which the throat symptoms predominate, a healthier state of faucial mucous membrane is obtained by using a gargle of chlorine water. If the child cannot gargle, a wash or spray may be used. Chlorine water can be quickly made by pouring strong hydrochloric acid (℔vij) on chlorate of potash (ʒij), and then adding water (ℨviij), which becomes impregnated with the nascent chlorine.

The irritative, acrid, nasal discharge occurring in the more malignant cases is distressing. I find nothing more soothing than irrigating the nostrils and inferior meatus with a solution of carbonate of soda (gr.x to ʒj).

There is yet another point in the treatment of scarlet fever, be it ever so mild, which requires attention and consideration. Deafness frequently results from scarlet fever. Even in mild cases a septic inflammatory process may travel up the Eustachian tube, and so disorganise the tympanum; hence, I hold it to be the duty of every practitioner to guard against this catastrophe. Not only is there the immediate danger of loss of an important special organ, but there is the remote risk, it may be years after, of pyæmia or of suppurative meningitis spreading from an infected tympanum, which has been forgotten or overlooked. So far as my experience goes, warm boracic acid gargles, or better still, the same drug in the form of warm lotions to the pharynx and Eustachian tube, give the most favourable results.

Diphtheria.—The supposition that membranous croup and diphtheria are different diseases is, in my opinion, erroneous. So long as there is false membrane the case is one of diphtheria, although undoubtedly we may see cases of diphtheria in which the membrane is not evident. The exciting cause is proved beyond doubt to be Lœffler's bacillus, but other factors must be at work before this bacillus can grow and propagate; among such factors appear to be the low standard of health which obtains in children attending overcrowded Board-schools, and the catarrhal throats which we see result so often from the inhalation of sewer-gas.

In the treatment of diphtheria, isolation is of course imperative, and although we naturally direct most of our energies to the local manifestations of the disease, the constitutional symptoms are even more important.

Firstly, as regards local treatment one important thing to remember is, that all gargles and washes should be warmed. Various drugs have been used for their antiseptic qualities. Chlorine appears to be useful in many cases, and the same preparation can be used as I described to you in the treatment of scarlet fever. Sulphurous acid again is a remedy which is universally popular, and the results warrant its popularity. Again, I have seen happy terminations of cases in which the throat was swabbed four or five times daily with a diluted solution of perchloride of mercury. I generally apply a strong solution of salicylate of soda; it appears to have a wonderfully solvent effect upon the membrane. Papaïn is also a remedy which appears to act quite as efficaciously and as speedily. I have seen attempts made to forcibly detach the exudative membrane, but the sequel was not a happy one; a gag had to be employed, and I think the struggles of the child were in some way conducive to the ultimately fatal result by syncope. Then there comes the question of tracheotomy. I fully endorse Murchison's rule that tracheotomy should never be performed on an infant under twelve months of age, as it is seldom or never successful, and the shock of the operation is usually sufficient to destroy the life of the child, in an already exhausted condition. A much safer and more successful plan is to practise intubation when there are signs of mechanical obstruction in the air passages. The results published by my friends, Dr. Wallace Ord and Mr. Staveley, fully bear out this statement.

As regards the constitutional treatment, our main line should be to give a generous and stimulating diet. The child has to hold out against an exhausting disease, and it has often occurred to me that one of the reasons of the high mortality from diphtheria which obtains in hospitals is due to the fact that the patients that enter therein are usually children whose strength is not up to the normal standard.

During convalescence, palsies affecting the various voluntary muscles must be looked for and guarded against, those paralyses which affect the arms, soft palate and the ciliary muscles of the eye are not necessarily of serious import; but I

have now records of five cases in which terribly sudden death has occurred whilst the child was at play, or whilst straining at stool, or after a violent attack of vomiting, and in whom a previous mild attack of sore throat had been overlooked, or its diphtheritic character either not recognized, or its gravity unduly weighed.

The antitoxin treatment of diphtheria is a subject on which I am not yet in a position to speak; but every practitioner will agree that such treatment is based on scientific research. Possibly the time will come when every zymotic disease shall be combated by its antidotal bacillary process, and I imagine that the public will demand, in the face of the overwhelming evidence of scientific research, that stations shall be instituted at various convenient parts of a town, at which practitioners can procure with ease, and without great cost, culture-tubes with which the diagnosis of diphtheria can be assuredly and swiftly made. An attempt is already being made in this direction; but such philanthropy ought not to be dependent on the zeal of a private society, it should rest on the shoulders of the community.

Typhoid Fever.—In the treatment of this fever there are practically two schools of practitioners— the one which conducts its patients through the fever, doing nothing the while but observing symptoms and checking any marked diarrhoea; the other school which holds that active measures are imperative throughout the whole course of the fever. Probably, the man who steers a middle course, which is between a masterly inactivity on the one hand, and an over-active interference on the other, will have cause for most congratulation. Those who prescribe medicines during the course of the fever give dilute acids, such as five-drop doses of dilute hydrochloric acid, or they give saline mixtures, and wait the development of events. Dr. John Harley appears to be favourable to the administration, three or four times a day, of small doses of grey powder; and when I look into his results at St. Thomas's Hospital during the epidemics of seven or eight successive years, I can but be impressed with the wisdom of his treatment. Whether the mercurial preparation acts as a disinfectant on the diseased mucous membrane of the bowel, or whether it acts favourably by keeping the bowel free from an accumulative poison, I am not in a position to say. I can only speak to you of results which have been eminently

favourable, and which have been borne up by my own practice. I have no favourable experience of the so-called antiseptic treatment of the bowel by the administration of carbolic acid and other remedies which act in a similar way. It has always occurred to me that it is impossible to keep the intestine in a state of perfect asepsis, and I am convinced that where once the chain of antiseptic treatment is broken, our plans are at once frustrated. Nevertheless, the treatment has strong scientific grounds for its recommendation. In the treatment of hyperpyrexia, the graduated bath has many advocates. The patient should be lifted in a blanket, held at each corner by an attendant, and should then be immersed in a bath of a temperature of 100° F. This temperature is lowered, by the addition of cold water, to 60° F. in twenty minutes. Others, again, resort to cold packing. Of the two, I prefer the latter procedure. I have seen patients who have died after the cold bath treatment, and although I could not convince myself that the cold bath was conducive to the fatal result, still I have not failed to observe in every case that the lungs had a peculiar slaty blue colour and collapsed condition. At any rate, this alone would compel me to withhold my sanction from a graduated bath in any case which presented signs of embarrassment of the pulmonary circulation.

Another essential point in the treatment of typhoid fever is that of diet.

I observed, when recording the epidemics at St. Thomas's Hospital, during the autumns of seven years, that constipation was a marked symptom in a percentage of cases much higher than one would suppose from reading the description of this fever in our manuals and text-books. On inquiry each year among many of my friends who were in general practice, I found that constipation was a rare occurrence in their typhoid patients, and the conviction was forced upon my mind that constipation occurring in hospital practice was entirely related with the rigid milk dietary which is enforced, whilst in private practice this severity of dietary is often replaced by rules which are somewhat lax. I do not hold, therefore, that some epidemics of typhoid are characterised by constipation, and others by diarrhoea; the two conditions may be accounted for, on the one hand, by a purely milk dietary, on the other hand by a varied dietary which does not counteract the normal flux diagnostic of the disease. The question then arises

under what conditions should we check diarrhœa, and at what period should we overcome constipation. A good rule is to regard four evacuations in the day as the safe limit. A diarrhœa which is in excess of this requires medicinal treatment. Opium or morphia may be given by the mouth, but preferably in conjunction with starch enema by the bowel.

In extreme cases only do I resort to the use of the mineral acids.

Those cases which are prolonged, and in which death is threatened by exhaustion, require alcohol in large doses. Twelve ounces of brandy or more may be allowed in twenty-four hours in desperate cases. Musk is also a most valuable stimulant; but I place it in a position secondary to alcohol. As regards constipation nothing is safer and nothing is more effectual than a warm water enema to which a little olive-oil has been added. There is one point which I wish to impress on your minds, and it is this,—that in the constipation which is occasionally found in typhoid fever, either a relapse or a recrudescence or a fresh rise of temperature nearly always follows the administration of a laxative or of an enema given to combat this condition.

Occasionally it is our experience to come across a case in which that terrible complication, perforation of the bowel, has happened, and we are called in consultation to see a case which is apparently hopeless. I think I have seen only one case recover in which perforation of the bowel had undoubtedly taken place, if we can place any reliance on signs which are supposed to be diagnostic. The other cases of perforation have all died. But I strenuously hold that when once the signs of perforation are undoubted, the patient must not be allowed to perish without some attempt being made by opening the abdomen to effect a cure by surgical means. These days of antiseptic surgery hold out hopes of brilliant results.

One practical point in treatment during convalescence I would wish to impress upon your memory. I have seen many cases of relapse date from the day in which bread was placed upon the diet sheet. The bowel appears to resent the administration of bread in any form, and this is an experience which will be confirmed to you when you ask the opinion of any intelligent nurse who has attended many cases of typhoid fever.

Lastly, on the point of prognosis, does the temperature give us any guide as to the probable issue of the case? I think it does. Those cases in which the intermissions are regular, and in which the highest point of the curve does not exceed 104° F. may be regarded as favourable cases, and in which the mortality is low. But a temperature of 105° F. is a dangerous limit. In the series of seven years which I have previously spoken to you about, which included 400 cases, I found that only four cases recovered in which a temperature of 105° F. had been recorded. This high reading might have occurred during the primary fever, or it might have occurred during the relapse, but whenever it occurred the termination of the case, with these four exceptions, was always fatal.

Whooping-Cough.—The frequency of this disease both in hospital and in private practice is my excuse for detaining you a few minutes longer. The death-rate from whooping-cough is, as you know, exceedingly high. I am of opinion that this disease ought to be included in the list of fevers which have to be notified under the special Act of Parliament. It would appear to be endemic in London and the suburbs, and for the last eight years I can safely say that not a week has elapsed without one or more cases presenting themselves at my out-patient room. I think some other disease might with advantage be struck out of the notification list and whooping-cough substituted. As regards the treatment, the first indication is to check catarrh of the respiratory system. A child with laryngeal and bronchial catarrh is in a favourable condition to receive and develop whooping-cough. We see this in cases with prolonged coughs due to bronchial catarrh and in which eventually the typical whoop is developed; and although we are not yet decided upon the duration of the incubative period, still we must conclude that it is a long one, although hardly so long as the preparatory catarrhal condition would in many instances suggest. My contention is that most children are, at one time or other, exposed to, and are recipients of, the specific bacillus, but that many only cultivate it under the influence of catarrh or other conditions favourable to its growth. Notwithstanding this, the first indication is to isolate the child who is undoubtedly suffering from the disease, and also to check, and to check speedily, if possible, any tendency to bronchial inflammation in any other child resident in the same house. When the disease is established without doubt, what is to be

our remedy? Many drugs have been tried and vigorously advocated; the list includes bromides, belladonna, conium, hydrocyanic acid, chloral, stramonium, oxide of zinc, and many others; but none of these is more useful in its action than the other—at least so far as my experience goes. The atmosphere which is impregnated by the refuse from gas retorts is vulgarly supposed to be useful, probably on account of its antiseptic powers; but of this we have no actual proof. I think an abundance of fresh air is a much more powerful antiseptic. But after all the treatment of the disease is indicated by the complications. If I were to write a list of the complications I should find them all, or most of them, included in one great category, viz., those which attend, and which are due to, the violence of the expiratory efforts. Thus we find vomiting, diarrhœa, prolapsus ani, hernia, and incontinence of urine. These are the main complications of the gastro-intestinal canal and urinary tract; in the lungs, the chief are emphysema and hæmoptysis; those of the circular system are represented by temporary or even permanent enlargement of the right heart, by epistaxis, by black eyes, and other forms of hæmorrhage. Now all these conditions yield to the internal administration of morphia. It is a commonly accepted doctrine that children, and especially infants, must be debarred from the good effects of this magnificent drug, but I think it is a mistaken notion. The dose of morphia can be regulated to suit the requirements of the youngest infant, and I hold that the drug is not contraindicated in young life. I am in the habit, therefore, of following Henoch's plan of prescribing morphia in infinitesimal doses, three or four times a day, until such period when the paroxysms shall have diminished in severity and frequency. I find the following prescription useful: Morphia gr.$\frac{1}{80}$; oxymel of squills $\mathfrak{m}$ 10; water to a drachm. In babies under twelve months old, I restrict the dose to gr.$\frac{1}{160}$; whilst to sturdy children of 2 and 3 years of age, I may increase the dose to $\frac{1}{30}$, or even $\frac{1}{25}$ of a grain. At the same time, I must admit that it is necessary to have the child under frequent observation, as some children respond to the drug so much more readily than others. But I think you will be satisfied with the results of this treatment, when you find the cough instead of running four or six weeks, has its duration cut down to half the time. Diet also is a most important factor. The meals must be small, yet frequent; and the food

should consist of constituents which are light and easily digested. A large meal is rejected at the next paroxysm, and the child is exhausted by the active vomiting. Milk, eggs, and farinaceous puddings, should be the only foods allowed. Butchers' meat is in my experience injurious, and even if permitted, it should take the form of weak beef-tea given in small quantities every hour.

A CLINICAL LECTURE
ON
MALIGNANT STRICTURE OF THE ŒSOPHAGUS.

Delivered at St. Bartholomew's Hospital, Nov. 21, 1894,

By H. T. BUTLIN, F.R.C.S., D.C.L.,

Surgeon to the Hospital.

GENTLEMEN,—Let me say, to begin with, that of all the symptoms of "malignant stricture of the œsophagus," I believe there is none more important than dysphagia; in the first place, because it is almost always the first symptom of which the patient complains, and secondly, because it is almost always the most constant of all the symptoms, with little liability to vary either in seat or intensity. As it is of so much importance it will be well to consider it not only as a symptom of malignant disease of the œsophagus, but also as a symptom of other troubles. In the first place, one has to separate in one's mind two kinds of dysphagia— dysphagia which consists of pain, and dysphagia which consists merely of difficulty in getting things down. Dysphagia which consists in pain is seldom of so serious import, and is much less common than when it consists in difficulty in swallowing. In what I have to say during this lecture I beg you to understand I refer almost invariably to the difficulty of swallowing, and not to the pain. Of course, there are people with malignant disease of the œsophagus, who suffer a good deal of pain in addition to difficulty, but pain in swallowing is comparatively an infrequent symptom and may be looked upon as an exception to the general rule. When a patient complains of dysphagia, the first thing necessary to take into account is whether he is under or over 40 years of age; that being a kind of general dividing age. Supposing a patient under 40 comes with dysphagia, I think I may say,

roughly speaking, that the chances are ten to one that there is no serious trouble—mind, I am not speaking of pain in swallowing. If, on the other hand, a patient who is over that age complains of difficulty in swallowing, the chances are quite ten to one that there is some serious mischief to account for this difficulty. People who are under 40—quite young people—sometimes suffer from difficulty in swallowing, and the difficulty may depend upon pharyngeal granulations. Or, the difficulty in swallowing may be due to muscular or spasmodic stricture, or to that rare condition, fibrous stricture, which is usually dependent upon some old ulceration in the œsophagus, and that very often again depends upon the swallowing of something that has burned or destroyed the membrane of the œsophagus. Of tuberculosis as a cause of dysphagia I shall not speak to-day, because, although that is a common cause of difficulty in swallowing, the difficulty is generally very painful.

When a patient complains of difficulty in swallowing, and is more than 40, I judge of the gravity of the case partly by the appearance of the patient and partly by the replies to a certain set of questions which I put. In the first place, let me say that persons who suffer from malignant disease of the œsophagus are generally males; perhaps one in six is a female. I have two patients here,* one having been treated for some time; he is wearing a tube at the present moment, which Dr. Thorne is going to change for him presently, in order to show you what kind of tube it is, and how it is changed.

The first question I ask is, " *How long have you been suffering?* " One patient says it is " since last Christmas—nearly a year," and the other patient, who is 44 years of age, has been suffering from difficulty of swallowing for about three months. The second question I ask is *whether they have lost much weight;* and this man will tell you that he formerly weighed something over eleven stone, and at the present time does not weigh quite nine, while the other has lost flesh very quickly indeed; he formerly weighed ten stone and now weighs seven stone, an enormous loss of weight in a very short space of time. The third question I ask is whether *the difficulty in swallowing has increased very rapidly.* One of these patients tells me he only takes arrowroot and beef-tea now, and the other says he can take no solid

* I am much indebted to my colleagues, Dr. Gee and Dr. Norman Moore, for the permission to show these patients.

food at all. That is most invariably the case with a patient suffering from malignant disease, in the course of a few weeks or months. Other questions are as to the *variability of the dysphagia*—as to whether it varies from day to day, or from week to week, and you will find, although a patient may say that he swallows better on one day than on another, still there has been rapid progress in the increase of the difficulty on the whole, judging from week to week. The difficulty is worse this week than last week, and was worse last week than the week before. And the seat of the difficulty is not liable to variation as is the seat of difficulty in spasmodic stricture, or the seat of difficulty in a patient with pharyngeal granulations. A patient almost invariably places his finger over one particular part of the course of the œsophagus and tells you the difficulty is there. The part of the course, however, at which the difficulty appears to him to be, although it is constant, is not by any means a certain index to the exact part affected. For instance, a patient with disease at the level of the cricoid cartilage will sometimes declare that his difficulty is much further down than it really is —only it does not vary, it is not sometimes here and sometimes there; he feels it almost invariably at the same point. I do not say there are no exceptions to this, but you may very fairly rely upon the rule. The last of the important questions I ask is *whether the patient spits up anything.* These patients spit up quantities of " phlegm," very often as much as a patient with tuberculosis, and large quantities of frothy fluid, which is a mixture of mucus and saliva, and very likely some food. The quantity of the frothy fluid has sometimes quite surprised me—it is enormous. Then I look for other evidence, and ask, for instance, whether the patient has ever brought up any *blood*, or whether he has brought up any *actual fragments* which were not fragments of food, and to that question the reply is very variable. On the whole it is rare for a patient with malignant disease of the œsophagus, except in the latest stages of the disease, to bring up blood, and it is equally rare to bring up fragments of the disease, although they may sometimes be brought up in the eye of a catheter which is passed down, or in one of the regular œsophagus tubes.

Some patients suffer from cough which may be very troublesome indeed. I speak now of the cough which depends upon direct irritation of the larynx, or trachea, by the malignant disease, or

which depends on the involvement of some of the nerves of the larynx ; this is much more frequent when the disease is situated high up in the œsophagus. Another sign is *hoarseness* of voice, rare when the stricture is situated low down, but by no means uncommon when it is in the upper third of the œsophagus. When these symptoms are present you may find a very important sign, *paralysis of one of the vocal cords.* A patient may complain of dysphagia and may have a certain amount of cough, and the voice may be very unsteady. The right vocal cord you find is paralysed, and these signs put together are very important indeed, because the hoarseness and the unsteadiness of the voice may depend upon the paralysis of the cord, and the paralysis of the cord may very likely depend on the cause which produces difficulty in swallowing —malignant disease, in nine cases out of ten.

Then, again, there are some patients who have *enlarged glands* on one or both sides of the neck, and although this is by no means a constant symptom, and, indeed, very frequently is absent, it is, when present, a very important symptom. Where the disease is situated in the lower half of the œsophagus the cervical glands are not affected, but when it is situated in the upper third, and particularly in the posterior plate of the cricoid, enlargement of the glands is by no means uncommon. I have seen extraordinary enlargement of the glands in some very unusual cases. Here is a sketch of a man who was in the hospital some time ago, who had a large mass in the neck. A drawing was taken of him to show what he exhibited in a marked degree— the signs of paralysis of the sympathetic nerve on the right side owing to the implication of the nerve in this mass. Those of you who are acquainted with this paralysis will know how rare it is to see a case of paralysis of the sympathetic nerve due to enlarged cervical glands. I had not the least notion that this patient had any serious disease of the œsophagus, because although he had this huge mass in the neck, there was but little difficulty in getting down his food—at no time any serious difficulty. It was only on examination after death that we discovered that he had malignant stricture of the œsophagus, and that the tumour in the neck was secondary to the malignant disease of the œsophagus. Such a case you may look upon as quite exceptional.

Having obtained answers to these different questions, I naturally proceed to a further examination of the patient, and one of the most important parts of such an examination is the passage of a tube or bougie. The passage of a bougie is an operation I always dislike to perform. It is very disagreeable to the patient unless he has been trained to it, and as I am often called upon to make the first attempt, I find it very disagreeable. In order to render it less distressing I almost invariably spray a small quantity of cocaine— 10 per cent. solution—down the back of the throat. It does not much matter which of several instruments is used, but I think the bulbous bougies are the easiest to pass. The patient sits in a chair, and the operation is conducted as you will see Dr. Thorne conduct it presently. He puts his finger into the mouth, holds the tongue down and then passes the instrument. The first point at which it hitches is almost invariably—indeed so invariably that you may look upon it as a constant difficulty— the posterior margin of the cricoid plate, in fact, on the back of the larynx, some six or seven inches from the teeth. Frequently I have patients brought to me with malignant disease of the œsophagus in whom an attempt has been made to pass some kind of bougie. The instrument has been arrested seven inches from the teeth, and I am told the stricture of the œsophagus is there. I am obliged to regard the statement with suspicion ; for as a matter of fact the œsophagus does not begin for about eight inches from the teeth, and the instrument has seldom passed out of the lower part of the pharynx. The way in which to overcome the difficulty is to pass the bougie down until it strikes against the posterior part of the larynx, and then to direct the patient to close his mouth, but not to bite upon the bougie. The instrument should be held there for a minute or two until he has recovered from the discomfort. He should then be told to swallow, and just at the very moment of swallowing, it should be passed on, when it readily enters the œsophagus. From this point it should pass smoothly and easily into the stomach. And you must bear in mind that the entrance to the stomach is probably at least $13\frac{1}{2}$ inches or 14 inches from the teeth, and may be as much as 16 inches ; in fact, I should not be sure if I passed a tube for only 16 inches, that it had passed into the stomach. It is quite possible it might have been arrested by a malignant stricture at the cardiac orifice. When the bougie comes to a stop at any point short of the stomach, it is pretty clear that there is a distinct stricture at that point ; and supposing the patient has all the symptoms of which I have spoken, you

may be as certain as possible that the disease is malignant disease; practically, when you have got thus far, the diagnosis is almost as complete as you can make it.

There are some other methods of diagnosis, and one of these is auscultation. I have tried auscultation. If a stethoscope is placed on the back over the seat of the stricture, you are supposed to hear a peculiar grating sound. I have generally heard the grating sound, but it seems to me that I hear almost as much grating in a person who swallows perfectly well as in a person who has a stricture. I have never been able to distinguish sufficiently clearly between the gratings which belong to proper swallowing and the gratings which belong to improper or stricture swallowing. I should not be surprised if one might attain to considerable skill in distinguishing between these grating sounds, but I think it would take a great deal more practice than I have ever had. Of the œsophagoscope I have had no experience.

Well, by the questions and methods I have mentioned, one can, as a rule, arrive at a very certain diagnosis of malignant disease of the œsophagus, and it is very rarely indeed that a mistake is made. On the other hand, it must always be understood that of a disease which you cannot see or feel—a disease so situated that you cannot get at it, and can only judge of it by a number of different signs, most of which are described by the patient—it is absolutely impossible to form in all instances a perfectly certain diagnosis. I have been mistaken several times where I thought the diagnosis was as clear as it could be, and I have known very singular mistakes to happen.

I will point out some of the diseases or troubles for which malignant disease of the œsophagus may be mistaken. In the first place, there is always the question of an *aneurysm of the aorta*, or of one of the large vessels in the chest, which may press upon the œsophagus at some point, and I advise you to be most careful in the passage of an instrument if the stricture appears to be from 8 in. to 11 in. from the teeth, because within those three inches there may be pressure on the œsophagus by an aneurysm. There are a good many cases on record in which the passage of an instrument has resulted in instant death from damage done to an aneurysm of the aorta, which lay across the œsophagus. Of course, in addition

to the symptoms of stricture, the patient may suffer from attacks of giddiness or breathlessness, or other symptoms which, even if the physical signs of aneurysm of the aorta are not by any means decisive, may be sufficient to warn you that you have to deal with a very different disease from malignant disease of the œsophagus. So I merely give it as a general rule that in all strictures or difficulties of swallowing which appear to be within from 8 in. to 11 in. from the teeth, you must have in mind the possibility of an aneurysm of the aorta, or of one of the large vessels, and must, therefore, exercise great care in the passage of an instrument. Then there is the possibility that the disease may be a *simple or fibrous stricture*, a disease of which we know very little, unless it results from some injury to the œsophagus, such as is produced by the swallowing of some corrosive fluid.

The diagnosis between this disease and cancer is rarely difficult, but I had under my care, about a year and a half ago, a gentleman whose case presented great difficulties in diagnosis. Professor Czerny and another celebrated German surgeon thought that he was suffering from malignant disease, and Dr. Durante, the chief surgeon in Rome, that he had a spasmodic stricture. Sir James Paget also thought the stricture might be spasmodic, although he would not offer a decided opinion upon its nature. I believe that it was really a fibrous stricture, for I was able to prove that it was not spasmodic. While the patient was in London he submitted to be placed very deeply under the influence of an anæsthetic, and as his stricture did not yield in the least degree, I take it that spasmodic stricture could be completely excluded. But it was never quite certain what the nature of the stricture was, for he died rather suddenly from an acute attack of pneumonia, in Rome, and no autopsy was made. I may say, however, that the pneumonia did not appear to be consequent upon the disease of the œsophagus, but was one of the regular attacks of pneumonia which are so common in Rome and some of the southern Italian cities.

Another disease I suppose I must put amongst those which may cause a difficulty of diagnosis is *rheumatism*. I should scarcely have thought it necessary to mention rheumatism if it had not happened that last July I had a woman in the hospital some 53 or 54 years of age, who, I thought, was suffering from malignant disease of the œsophagus. She was a deaf old woman, and

uncommonly stupid, and it was very difficult indeed to get any history from her. From what she said she had been suffering from some kind of discomfort in the throat for about a year, and on the 24th June began to experience much greater difficulty in swallowing. She was admitted into the hospital on the 3rd July. At that time I could not see any actual disease in the throat, but I could see that the right vocal cord was paralysed. I passed an instrument, and it seemed to come to a stop just below the cricoid cartilage, and I therefore, taking into consideration her age and the sudden increase in the difficulty in swallowing in a patient who had suffered from some trouble in her throat for a long time, came to the conclusion she was suffering from malignant disease of the œsophagus. I kept her in the hospital and gave her a spray of cocaine, and she was kept on fluid diet, which indeed was the only diet she could swallow. I wrote to her doctor to tell him that I was afraid she was suffering from malignant disease of the œsophagus, and as it was high up I was afraid I could not do much by means of a tube. The letter had hardly reached him when she became very much better, and when I examined her at the end of another week, I found her symptoms had almost passed away. She could swallow solids, the paralysis of the cord had disappeared, there was no tenderness, and she had neither pain nor difficulty in getting down any kind of food. I therefore came to the conclusion, as she had been subject to "rheumatics" for a long time, that in all probability her trouble was due to a sudden attack of rheumatic inflammation in the crico-arytenoid joint, and some consequent swelling a little lower down than I could actually see. But that is the only case I have met with in which there was sufficient reason to believe a patient was suffering from malignant disease when she was suffering from rheumatism.

There are some cases of very uncertain origin, and where one cannot tell what the nature of the disease is. I had a gentleman, some 60 years of age, under my care about twelve months ago. He told me he had experienced difficulty in swallowing for many months. He had lost about a stone in weight in a comparatively short time, and from his symptoms I feared that in all probability his disease was malignant. But he is still living, and in very much the same condition, and he can swallow just as well as he could then. He has gained flesh again, and as he has been suffering now for many years from some kind of stomach trouble it is quite possible he had an attack of chronic inflammation of the œsophagus about the cardiac orifice. But I do not know for certain what the exact nature of his disease is.

Now, Gentlemen, I can say what I did not like to say before these two poor men, that the prognosis, of course, in every case where a patient is suffering from malignant disease of the œsophagus is terribly bad. But very few people live more than a year with it, and a great number die within a few months—sometimes within two or three months—of the first appearance of the symptoms. Most of them die from what may be called marasmus—they get weaker and weaker and thinner and thinner until they · are absolutely exhausted. Both of the men we have seen to-day are miserably thin. They have lost three stone in weight, and are practically worn out, and you have heard them say they can only take fluid food. Although one of them has gained in weight since the tube has been passed, he does not weigh quite nine stone even now. Very few of them suffer from complete obstruction. Sometimes, however, within the last few days of the disease complete obstruction occurs, and the patient is absolutely unable to swallow any kind of food, solid or fluid. Many of them suffer from lung complication—pneumonia for instance, broncho-pneumonia, and the like, and in some cases a very troublesome complication is met with—perforation of the trachea. When I spoke of cough just now, I had in mind the cough produced by this complication, which is very distressing. I can give you a case which will show you exactly what may happen. I do not know how many of you may remember a man—a small farmer—who was in the hospital a few months ago, in Colston Ward, on account of difficulty in swallowing, due to malignant disease of the œsophagus. I passed an instrument—one of Symonds' tubes, which was kept down, and he did very well for a time, and was able to go about his farm, and superintend his men at work. After he left the hospital, he came up to see me once or twice at my house, and once he came in rather a hurry and told me he had brought up more blood than usual. He had been troubled with a very great cough and hoarseness, and when I inquired, I found that every drop of liquid he took produced a most violent cough, and the poor fellow coughed so violently in my room, that I was afraid he might die there. He was very anxious that I should pass some other kind of tube, or do something for him,

but I knew it was very dangerous to attempt to pass a tube for a man in that condition, and that no tube would be likely to relieve him; for the disease had ulcerated into the trachea or bronchus. Fluids pass down by the side of the tube, make their way into the air-passages, and produce violent irritation and cough. Patients in whom such perforation has taken place usually die in a few days, because in spite of the coughing, and getting rid of some of the fluids, a small quantity is sure to pass down, and it sets up that form of pneumonia to which the Germans have given the name of Schluckpneumonie ("swallowing pneumonia"). I have rarely known a patient to die of hæmorrhage from a stricture of the œsophagus, unless it was after the passage of a tube, so you may look upon that as infrequent.

Now comes the question of what can be done for these poor people, and I am sorry to say surgery does not hold out a very happy prospect of relief. I believe that the best way of treating all these malignant strictures, when it can be done, is by means of a tube; and I have various tubes to show you what may be used under different circumstances. Symonds' tubes are of different kinds and sizes.* They are funnel-shaped at the top, and are made of material which speedily softens *in situ*, so that it does not exercise an undue pressure in the interior of the œsophagus, and a patient wearing one of these is comparatively in comfort, and can swallow every kind of fluid. I have known patients wearing these tubes swallow jelly and food of similar consistence without any difficulty, and very rarely indeed does the tube become obstructed. You see how it is fastened in this particular case by a thread issuing from the corner of the mouth and fixed around the ear. I have more frequently fastened the thread to one of the teeth—particularly in the case of persons who are not hospital patients, because they do not like to go about with a thread proceeding from the mouth. Symonds' tube is applicable to strictures about the middle of the œsophagus; the neck of it lies in the stricture itself, and the funnel immediately above the stricture, and there is very little danger of its being swallowed, although I have known such a thing happen. But Symonds' tube, which I regard as the best of all such tubes, is rarely applicable to a

stricture much above or much below the middle of the œsophagus. If the stricture is high up, the funnel of the tube lies between the back of the larynx and the spine, and the patient cannot bear it in that situation; or, if the stricture is situated close to the cardiac orifice, the end of the tube must lie in the stomach, if you are fortunate enough to be able to pass it at all, and many patients will not put up with that. So really the use of Symonds' tubes is limited to strictures which are about 10 in. or 12 in. from the teeth.

In cases in which the stricture is high up, a tube may be used in the manner suggested by our excellent colleague, Mr. Berry. Mr. Berry wrote a very good article in the "St. Bartholomew's Hospital Reports," some years ago, on the use of railway catheters and soft tubes in the treatment of cancerous stricture of the œsophagus (vol. xx. p. 45). In the case of stricture high up, a long catgut is first passed, and over it a gum-elastic railroad catheter, which is kept in for a day or two, until the stricture is dilated to a certain point, when, in the place of the hard tube a soft tube is passed, and is fastened by a thread either to a tooth or round the ear in the same manner as Symonds' tube.

The treatment of malignant stricture within eleven and twelve inches of the teeth is comparatively a simple matter; although there are some patients who either will not, or cannot, endure the continual presence of a tube. Such people may be allowed to remain untreated locally, provided they can swallow a sufficient quantity of fluid food. Or the stricture may be dilated by the frequent passage of a tube, provided the tube is passed on a guide, and the greatest caution is observed.

But, what is to be done in the cases of intolerant patients who are more than half-starved, and in cases in which the stricture is situated at or near to the cardiac orifice of the stomach, and is practically impermeable? I regard these as the cases in which the operation of gastrostomy may very properly be performed. A successful gastrostomy must be regarded as a means of relieving the distress of hunger and of prolonging life; but let me advise you not to exhibit it to your patients or your patients' friends in too favourable a light; for both they and you may be woefully disappointed. To-day there is not time for me to speak of the operation, but I may do so in a future lecture.

Another operation, œsophagotomy, is really scarcely applicable to the treatment of stricture of

* Mr. Symonds' paper is published in the "British Medical Journal," 1889.

the œsophagus, although it may occasionally be performed for malignant disease of the pharynx.

And, the radical operation of œsophagectomy is practically unknown to me. It has only been performed a very few times, and is never likely to be frequently performed, on account of the difficulty of finding cases in which the disease is within reach of removal.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Erythema Multiforme—Questions as to its Nature.

A man, aged 25, sent by Dr. Vinrace, presented an excellent example of erythema multiforme on the backs of his hands. It had been present eight days. The patches, which were very numerous, were elevated, and somewhat like the wheals of urticaria, only much redder and with a slight depression in the centre. They varied in size from a split pea to a sixpence. The patient had a patch of erythema in front of his right knee, but it was larger and at the same time less raised than those on the hands. He stated that he had had a similar attack on his hands nearly two years ago, and that it passed completely off in a short time. He was liable to chilblains every summer and winter, and not unfrequently had bad colds, but did not recognize any special tendency to rheumatism. He was a tall man of rather feeble circulation.

Comments.—We must not, in the present day, be content with merely giving names to the phenomena which present themselves to us as " skin diseases," nor must we stop with mere descriptions of external appearances or records of progress. The problems before us concern the causes of what we see, and the mutual relationships between different type-forms of disease. To call such a case as this " erythema multiforme " is perfectly easy, and those who have once attentively inspected a good example of it can scarcely ever miss its recognition in future. The mere name also, I admit, brings with it very useful knowledge as to prognosis. We know that the eruption will fade away spontaneously after a short duration, and that it is not improbable that the patient will, with varying intervals, have other attacks. As regards therapeutics, I believe one may also say that we know that a long course of arsenic will be very useful in preventing the recurrence of the malady. There yet, however, remains the question as to its real nature and its alliances. First, I think we may assert that it is closely allied to other forms of eruption on the hands, which, like it, recur periodically, are of limited duration, and disappear spontaneously. Amongst these are some which are bullous or vesicating, and not erythematous at all. To this group belongs the malady which many years ago I described as cheiro-pompholyx. You would observe that I questioned this man closely as to his liability to catarrhal affections. This was done because there seems reason to suspect that in some cases these transitory eruptions on the extremities are really of a catarrhal nature, *i.e.*, that they occur in connection with the same causes and the same kind of general neurotic disturbance as a common cold in the head. Our patient, as you have heard, said that his attack this time had occurred during a very bad cold, but he did not remember anything on this point as to the former one. That these eruptions are in some sense neurotic, I do not feel any doubt. Most eruptions which are spontaneously curable and of limited duration are so, more especially those in which the development is always symmetrical. There is, however, another fallacy to be borne in mind, and that is, that they may possibly be due to some drug or article of diet which the patient has taken. They are indeed really not at all unlike the eruption which in some persons is caused by chloral, and which may occur exclusively on the hands. If, however, this be the case, the drug or dietetic poison acts probably through the nervous system, and not, as we may plausibly suspect to be the fact in urticaria, through the blood. Were it the latter the eruption would probably be much more general. Some have thought that erythema multiforme is a rheumatic manifestation, and there is considerable plausibility in the suggestion, but we must remember that rheumatism, when it occurs in connection with exposure to cold and damp, and is paroxysmal, is probably very closely allied to catarrh. I have recently been attending a young lady in whom attacks of a vesicating eruption on the hands recurred with great frequency and, as her friends thought, in connection with the ordinary

causes of catarrh. During a six months' course of arsenic she had been wholly free from it, although there had at times been slight threatenings.

Peculiar Eruption on Arms and Face, possibly a sequel of Ringworm.

A woman, aged 46, was the subject of a marginate eruption on her arms and face. It was very slightly marked, but quite definite when looked for. The margins of the patches were slightly raised and erythematous, the patches were very large, and appeared to have been produced by circles which had coalesced. To some slight extent the parts from which the eruption had receded appeared to have been bleached. She stated that she had had the eruption on and off for four years, that it had been much worse three or four months ago, and was now again getting well. On inquiry as to any ringworm in her family, she said that one of her children had suffered from it ten or twelve years ago. Dr. Barrett, who had brought the patient, had made a careful examination with the microscope, and could detect no fungus.

Two portraits were exhibited in connection with this case, both of them exhibiting very large patches on the chests of adults, which were more or less similar in character. In one of these, the precise nature of the malady had never been determined, as the microscope had always failed to detect fungus. In the other there was no doubt that the disease was common ringworm, and that it had been acquired by a mother who was attending to her children's heads.

Comments.—I do not think that this case can be classed as one of leucoderma. The bleaching is incomplete, and the margins of the patches are distinctly congested. The woman's statement that she once had a thick, red rash may, I think, be held to be conclusive. In spite of the negative evidence of the microscope, I am inclined to regard the disease as of parasitic origin, and probably due to the ringworm mould growing on an unfavourable soil. You may perhaps think it unreasonable to attribute it to contagion from her child's ringworm, since she has said that the latter was ten years ago. It is very possible, however, that she exaggerates this interval; indeed, her first statement was eight years, and we must remember that she herself has suffered for more than three. I believe that the ringworm fungus

often passes into a state of quiescence, possibly of what botanists call "resting spores." At any rate, a patient may be for a long period without any manifestations of its presence, and it may then again develop activity. Not long ago I treated a surgeon for a very peculiar form of ringworm on his buttocks. It was more than twelve years since he had left Burmah, and during that time he had been quite unaware that he was carrying the parasite about with him.

A Chronic Papillomatous Eruption on the Legs.

Dr. Corbet Fletcher, to whom we have been indebted for many interesting cases, has brought us to-day a patient who furnishes a good example of a disease which you will not find described in text-books, yet it is a very well-characterised one, and the instances of it differ only in degree of severity. Dr. Fletcher's patient is a man of 38, florid, and in robust health. On the front of both his legs there are rough, dry papillary patches. His right leg, on which the eruption has been present nearly four years, is in a much more advanced condition than his left, on which it has only recently made its appearance. The two furnish us an excellent opportunity for observing the early as well as the late phenomena.

We will take first, then, the one last attacked. On it you see simply some dry, scaly, ill-margined patches, at the base of which the skin is a little thickened, but scarcely at all congested. Near to them some of the hair follicles are distinctly enlarged and occupied by small dry plugs of sebum. The patches bear evidence of having been scratched, but they do not appear to have resented it much, for they are not in the least inflamed or sore. The man admits that they itch very much, and that he frequently scratches them at night. If we turn now to his other leg we see very conspicuous and peculiar changes. The patches are irregular in shape, but abruptly margined and so much thickened that they are raised, from a quarter to half an inch, above the level of the skin. They appear, so far as the naked eye can determine, to consist of enlarged papillæ, which have been welded together by interstitial growth and which are surmounted by dry epidermis in the process of exfoliation. They are, on their summits, of almost snowy whiteness, but their desquamation is branny, and not at all like the silvery

scales which characterise psoriasis. I have seen some dozens of examples of this malady and have recorded several. I used to call it papillary psoriasis and in some instances confused it with the acuminate form of lichen planus. It has, however, no real alliance with the latter disease, and perhaps only a remote one with the former. Its connection with dry eczema is probably much more close, and, as you have heard, the patient in our present case states that his mother suffered from eczema of the legs in connection with varicose veins. Without, however, seeking to associate with it any very definite name, let us endeavour to recognize clearly its clinical peculiarities. Like Bazin's malady, it is a disease of the legs, and the legs only. Further, it may be said that it is a disease usually of the fronts of the legs only, a statement which is not true of the Bazin cases. I am far from hinting that there is any connection between Bazin's ulcers and these cases, further than that they are both located by the structural peculiarities of the part attacked. This malady has probably nothing to do with scrofula. Its causes, we may plausibly suspect, are very simple. An hereditary tendency to eczema, some slight local irritation, resulting in congestion and irritability and, lastly, persistent scratching. We may suggest, further, that it occurs to patients who are in sound health and in whom there is but little tendency to suppuration or moist exudation. Were this not the case, precisely the same causes might result in a pustular inflammation, or in eczema rubrum, or in chronic œdema. It is only by taking into account all these various elements of causation, and allowing them to join in partnership, that we can gain a correct appreciation of the affection. Permit me now to add another item of hypothesis. All chronic inflammations are probably attended by the development of a material which is infective. Thus, in these patches, I have no doubt that one produces others, and that unless arrested by treatment the patches have a natural tendency, in virtue of their infectiveness, to spread. In this way I explain the development of the disease on the leg most recently attacked. The affection will not, however, leave the legs, and this constitutes one of its chief peculiarities, just as it does in Bazin's malady and in the condition which we know as "coffee stains on the legs." Although it may be thought that I have suggested an etiology sufficiently complicated there is yet one item which must not escape

mention. Very probably the patients who develop these patches are hereditarily prone in some slight degree to papillary overgrowth. We know that such tendencies do run in families. To complete my picture I have to add that this disease has nothing whatever to do with syphilis, that it does not occur in early life, and that it is met with more frequently in senile periods than in young adults. Its subjects are almost always in good health. It is intractable under treatment, incurable and steadily progressive unless treated. The measures to be adopted are those suited to other forms of what we may call papillomatosis. The overgrown papillæ are to be repressed by pressure and local applications. The latter may be used in considerable strength, and I have found the painting of them with creasote and the subsequent use of a Martin's bandage amongst the most useful.

Lichen Planus in an Early Stage.

The subject of this case was a stout, healthy man, an undertaker, who had been sent by Mr. Waren Tay. His eruption had been present six months, but was still restricted to the fronts of forearms, backs of hands, loins, and buttocks. His eruption was, in most parts, very florid, and the spots were very small. They were in some parts grouped, but in most scattered without any definite arrangement. On the fronts of the wrists they crossed transversely in bands.

Comments.—No example of this disease could better demonstrate its lichenoid character. The earliest stages in all gradations of the papules are well seen on the man's forearms. It is perfectly obvious that the eruption begins by minute papules which are formed about the orifices of the hair follicles. This is our anatomical definition of the word " lichen." In its later stages in lichen planus we see these little papules becoming confluent and merging themselves in larger ones, which become polished on the surface. This smooth polished top, the characteristic feature of lichen planus, is well seen in the present case, though on rather a small scale. No one who has once seen it can mistake the malady. Clinically, of lichen planus we may say that we know nothing whatever as to its cause. It occurs to perfectly healthy persons, is quite symmetrical in its development, and usually lasts in spite of all treatment for a certain time, and usually in the end disappears

completely. It may often be doubted whether the remedies used have any share in the cure, for it is certain that a tendency to spontaneous disappearance .is part of its nature. It is very important to take note of this, for it constitutes a very marked feature of essential difference from psoriasis, eczema, and other forms of chronic skin disease. You may confidently predict in lichen planus that the skin will, in the end, be restored to perfect health. The eruption, however severe, usually disappears and leaves no trace behind. Its subjects are, however, unfortunately liable, after a series of years, to have second or even third attacks. In its early stages the best remedy is antimony, but in the later ones' arsenic may be substituted.

Hypospadias, with supposed resemblance to a Female.

The subject of this case, a man aged 37, had his penis so buried between the testes that the latter at first sight looked somewhat like the labia of the female. On examination, however, it was easily ascertained that it was a case of hypospadias, in which the urethra opened in the perineum, and the body of the penis, which was small, was tied down. There was no pretence whatever for a mistake as to sex, yet the curious history was that the man had been christened " Mary," and brought up as a girl. He had been a pupil-teacher, a housemaid, and a dressmaker. From the first vocation he had been dismissed for his fondness for romping with boys, and from the others on account of suspicions in the opposite direction. Ultimately a medical examination was made, and at the age of 17 he was put into male dress. Since then he had developed a strong moustache, and assumed masculine features about which there could be no mistake.

Comments. — There are, of course, cases of mixed sex or imperfect hermaphrodites, in which some of the organs pertaining to one sex are but partially suppressed, and those of the other incompletely developed. It is indeed true in a certain sense that all beings, whether accounted male or female, partake in a greater or less degree of the attributes of the other sex. It is a question of more or less complete suppression, and the suppression is never absolute, as is shown by the retention of the mammary glands in the male. In the case before us, however, we have simply a defection in the development of one of the male

organs. There is no reason to suspect that any of the female organs are less completely suppressed than is usual in the male. A defect in the development of the penis is the sole abnormality. If any proper professional care had been taken in the boy's infancy, such a blunder as taking him for a girl ought never to have been committed. As regards the hypospadias, it is a deformity which presents itself in the most various degrees, and fortunately the more common ones are very slight. We had here not long ago, as some may remember, two brothers in whom the very slightest form, entailing no inconvenience whatever, was present. The present case is an example of one of the most severe. It is a deplorable condition. The diminutive penis is tied down so as to entirely prevent intercourse, and the orifice of the urethra is in a position which entails the utmost inconvenience in micturition. The man's testes, although placed high up on each side of the root of the penis, are of almost normal size, and he is, so far as they are concerned, perfectly virile. I am sorry to say that I do not see what can be done for his relief. It would be quite impossible either to liberate the penis or to prolong the urethra.

A Case of Unilateral Sweating of the Face.

This patient, brought by Dr. Sequeira of Aldgate, was a woman aged 64 in tolerably good health, who had for four years suffered from excessive sweating on the right side of her face and scalp. The sweating varied much at different times, but was often sufficient to be a great annoyance to her, soaking her hair and any articles of head-dress that came in contact with it, and at night wetting her pillow. She said that it was increased by any kind of excitement, and whenever she felt out of health. The whole of that side of her face, and more especially the eyelids and near to the inner canthus, showed small watery blebs which were persistent and did not break. The largest of these were as big as peas. Most of them were quite transparent, and they exactly resembled the little cysts sometimes seen in xanthelasma. The xanthelasma regions were specially affected by them. A few of these sweat cysts were present on the lower eyelids of the other side, but they were much fewer in number. During the examination it was easily seen that the one half of the patient's forehead became moist with perspiration,

whilst the other was dry. Dr. Sequeira had examined the sweat and found that it had an acid reaction and contained chlorides. The patient had been at times greatly troubled by excessive flow of saliva, but she had not noticed that it was more on one side than on the other. She also complained that she had great irritation of the right half of the tongue, and that she had experienced, for a period of two years, complete loss of taste. From this, however, she had recovered. There was a history that in early life she had been liable to very severe attacks of hemicrania, not attended by sickness, but often sufficiently severe to quite incapacitate her. The pain in these attacks had always been on the right side. She considered herself gouty, but had never had a definite attack in the toe. She was the mother of a large family, and, excepting the attacks of headache, had enjoyed fair health.

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On the causes of Lichen Marginatus on the Chest and Back.

We are indebted to Dr. C. Fletcher for bringing us a case, which, although not rare, is a very well-characterised example of a very peculiar skin disease. The patient, as you see, is a robust man. His only affection consists of the development of little circles of a lichenoid eruption in the middle of his chest, and the middle of the back. These rings are but few of them quite circular, most of them being made up of two, three or more which have coalesced. Their margins are however,—I speak now of what we see on his chest,—quite abrupt, and they consist of a number of very minute papules. The area which is enclosed shows no papules, but is quite smooth. On the man's back the eruption is so extensive, that the little rings have run into each other almost everywhere, and the whole surface is covered. There is no tendency to the formation of pustules or crusts of any kind, and little or no desquamation. What we call the lichenoid papules are so small that it requires careful looking to see them, but they are certainly there. We may call the disease lichen annulare, or lichen circumscriptum, or lichen marginatum. The name does not matter much. What concerns us most to know, is that the disease has probably more real affinity to ringworm than to any other of the varieties of lichen, and that it is a purely local affection. I do not know that anyone has as yet demonstrated the presence of any parasite, vegetable or otherwise; but the disease originates and spreads exactly like a parasitic malady. It is wholly distinct from tinea versicolor, to which indeed it bears scarcely a superficial resemblance.

Like it, however, it spreads slowly from a single spot, persists for many years, and rarely or never transgresses its accustomed boundaries. If you see these rings on a patient's chest, they are always near its middle, and you may be sure that they are present also in the middle of the back. If you see them in the middle of the back, you may be sure that they are present on the chest, and you may also feel tolerably confident that they are not to be found anywhere else. Of another thing you may also feel assured; it is that the patient wears flannel next his skin, and that he usually sleeps in the same vest that he wears by day. By altering these conditions, the disease is quickly cured. You have but to instruct the patient to stitch a piece of lint inside his vest, back and front, and to cover it with white precipitate ointment, and the rings will soon disappear. It remains for some future investigator, and probably it would be no very difficult feat, to demonstrate to us the precise cause of this malady.

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THERAPEUTICAL NOTES AND FORMULÆ.

Malakine in Rheumatism.—Dose in acute articular form, ℨiss in twenty-four hours. As much as ℨiiss may be given without danger, if doses be sufficiently divided. Increases diuresis, facilitates elimination of uric acid, and lowers temperature. No untoward effects.—(*Loire Méd.*)

Thymol in Toothache.—Dr. Hartmann has employed thymol in toothache from hollow teeth in place of arsenious acid. He fills the cavity of the tooth with a tuft of cotton, on which a few crumbs of thymol have been sprinkled. It does not irritate the mucous membrane of the mouth much, and it is easily removed by rinsing the mouth with water. If a rapid action is desired, let the patient rinse the mouth often with warm water, in order to facilitate the solution of the drug. It never increases the pain at first, as arsenic does, and is not poisonous.

(*Deut. Med. Woch.*)

Depilatory for Surgical Operations.—To remove hair from scalp, peri-anal, or scrotal region, without using razor, cut hair off with scissors, then apply paste made of sulphohydrate of calcium, mixed with a little water. Make layer one millemetre thick. No pain or erythema. In less than ten minutes hair can be rubbed or washed off, to grow again in several days.—(*Médecine Moderne.*)

Some Uses of Belladonna.—Dr. W. Murray has obtained excellent results with belladonna in renal colic, painful defæcation in women with utero-ovarian affection, as well as in intestinal occlusion and typhlitis. In renal colic he administers the tincture in doses of thirty to forty drops, repeated every two to three hours until a slight delirium sets in. Its administration until slight symptoms of poisoning are observed, is a *sine quâ non* in treatment, and it will soon be followed by a disappearance of the pains and a passage of the stone. In the painful defæcation of utero-ovarian affection he employs the following suppository:

℞   Mercurial ointment   ...   gr.ij
     Extract of belladonna ...   gr.j
     Subnitrate of bismuth ...   gr.iiss
     Cacao butter, q. s.

     Sufficient for one suppository. Introduce
         two per diem.

Bismuth is added in order to render its action on the intestines less irritating. In intestinal obstruction he advises the application of a square compress to the abdomen, upon which extract of belladonna in vaseline has been spread.

(*La Semaine Médicale.*)

Cancer of the Stomach.—Chlorate of sodium ʒii–iv daily, beginning with ʒij, increasing amount until vomiting and hæmatemesis have ceased ; mix drug with water f ʒ3¼, giving teaspoonful doses. More than ʒiv in twenty-four hours not to be given, lest bulbar accidents occur. Albuminuria a contra-indication.—(*Gaz. Méd. de Strasbourg.*)

Excoriations in Children. — The following ointment is spoken highly of in the excoriations of children:

℞   Salicylic acid   ...   ...   gr.viij
     Subnitrate of bismuth ...   ʒij
     Starch   ...   .   ʒiss
     Rose ointment ...   ...   ʒj

(*L'Union Médicale.*)

REVIEWS.

Fibroid Phthisis. By the late Sir ANDREW CLARK and Drs. HADLEY and CHAPLIN. (Griffin & Co.)   *Published at 21s. nett.*

We have read this book through carefully from cover to cover, and feel that we have been most pleasantly beguiled into a thorough study of an affection which has hitherto attracted only occasional notice. The chapter on the history of the disease is most interesting and proves that from time to time cases of fibroid disease of the lung have been observed, but have been very differently described by the observers, who, without knowledge of the bacillus of tubercle, have been much divided as to what was and what was not tubercular. The work before us should fix very definitely for all time the exact position which fibrosis occupies in destructive diseases of the lung, whether those be tubercular — *i.e.*, bacillary—or non-tubercular. It is very well written, and owes its chief charm to the exceedingly clear manner in which the facts are placed before us; whether the disease is sufficiently common to make the book a widely read one is open to doubt, but there can be no hesitation in saying that no one can consider himself an authority on disease of the lungs without having read it. The coloured plates are beautiful reproductions of the naked eye anatomy of fibroid lungs, but there can be no doubt that they have added very materially to the expense of the work.

Diseases of the Breast. By W. ROGER WILLIAMS. (Bale & Sons.)

Published at 21s.

The opening chapters of this book give a fairly full account of the development and natural history of the breast, including the variations—amazia, polymazia, hypertrophy, etc. The histology of the gland is next described, and then the bulk of the book is mainly composed of statistical details of cancer and other tumours in general, and of those of the breast in particular. Mr. Williams displays considerable skill in the manipulation of his figures, but figures will prove anything, and his conclusions are often at serious variance with those of most writers on the subject. To those in search of numerical details the book may recommend itself, but the absence of clinical features render it disappointing.

A CLINICAL LECTURE

ON A CASE OF

SYPHILIS SIMULATING VARIOLA.

Delivered at Guy's Hospital, Nov. 17, 1894,

By P. H. PYE-SMITH, M.D., F.R.S.,

Physician to the Hospital.

A PATIENT, 19 years of age, but looking older, was admitted under my care into Philip Ward on the 24th of October, 1894. He complained of pains in the limbs, and there was slight, but unmistakable, effusion into one knee-joint. There was only slight fever (100° Fahr.), and, as he gave a history of gonorrhœa, the case was naturally put down as synovitis due to that cause.

Here let me remark, in passing, on the fallacies of what we call a patient's history. When accurate and trustworthy, as the records of a good ward clerk, or the account of a family doctor who has attended the patient in all his illnesses from birth, a history is invaluable, and in some cases makes easy a diagnosis which would be otherwise impossible. A mother's history of her child's illness is also valuable when it has been patiently brought out by cross-questioning, and the facts disentangled from statements of what she thought, and what the nurse thought, and what remedies they used or meant to use, and why. But histories such as we get from hospital patients, and even from educated persons, are most uncertain. As a rule men say they have never been ill before in their lives until careful questioning brings out a long series of ailments which were not counted, because some were children's diseases, and others accidental, and others his own fault; while women will often exaggerate every cold into influenza, a cough into bronchitis, indigestion into ulcerated stomach, and headache into congestion of the brain. If you have much to do with assurance practice you will find how difficult it is to obtain plain answers to plain questions. People's memories are bad, unpleasant facts are easily forgotten, and none of us can regard our own cases from an impartial standpoint.

These difficulties are all increased when we inquire concerning venereal diseases. Those who are not ashamed of vice are almost always ashamed of its punishment, and apart from deliberate falsehood you will be met with many shifts and verbal equivocations. Practically, I advise you to use your eyes rather than your ears. If you find adhesions of the iris, scars on the fauces or the tongue, enlarged lymph glands, pigmented scars, or sarcocele, you need ask no questions. Always remember that it is possible for a man, and not infrequent for a wife, to have syphilis innocently. You will also do well to be satisfied with an admission of venereal disease generally, without expecting your patient to make a diagnosis in which the most experienced surgeons sometimes fail, between a soft and a Hunterian chancre. Syphilis is a specific fever, not invariously conveyed by sexual intercourse. Venereal disease includes syphilis, soft chancre, gonorrhœa, and, as I have more than once seen, scabies.

In our present case actual inspection showed that there was no gleet remaining, and that there were the scars of suppurating buboes in the groin, and repeated cross-examination at last showed that the urethritis had ceased more than a year ago, while the chancre had appeared last June.

The synovitis quickly subsided — a sufficient proof that it was not gonorrhœal. The moderate pyrexia continued and increased to 102.4° on the fourth day after admission. A few pustules were then noticed on the face. These might have passed for acne, the commonest facial eruption at this patient's age; but the distribution was too irregular for this, and the comedones which mark the glandular origin of acne were absent. Next day a continued rise of temperature and the spread of the eruption beyond the limits of acne prevented the least experienced from making such a mistake. On the fifth day after his admission there was an abundant papular and pustular eruption over the face, arms, and hands, and more sparingly on the trunk, buttocks, and thighs. With this the temperature had risen to 103° F. without quinsy, pneumonia, or any other local inflammation showing itself.

A pustular eruption with pyrexia should always

make one think of small-pox. In such a case look first for vaccination marks. We found four pitted white depressed scars on the left arm. The patient had, therefore, been well vaccinated in infancy, and had scarcely attained manhood, so that if variola were present it was modified variola. This, in fact, is what you will usually meet with in this country, and it makes diagnosis the more difficult because we do not know how far the protective influence of the attenuated virus may modify the symptoms. The papular and pustular form of the eruption, the fact that some of the pustules showed a marked central depression, and the distribution chiefly on the face, freely on the upper extremities, and travelling down over the trunk to the thighs and legs successively—all this closely resembled variola. There was also continued fever and a thickly furred tongue, while the muscular pains which preceded the eruption seemed to confirm this diagnosis. Even synovitis sometimes occurs in variola, though usually later in its course.

On the other hand the symptoms seemed too severe with his four good vaccination marks. He denied having had lumbago or vomiting, or even headache at the outset of his illness. A negative statement, however, of this kind will not bear much weight. I once made a mortifying mistake in the case of a man-servant who absolutely denied these symptoms, and asserted that he was quite well until a few pustules appeared, both statements being untrue. Nor was the absence of a previous case from which our patient might have caught the disease any help. I once saw variola in a child which had not left its nursery for several weeks before, and the parents could not believe that it could have caught it. It afterwards came out that a nurse-girl, who immediately left for fear of catching the disease from the child, was really afraid because two of her own family were ill at home with modified small-pox.

Much more important than the history was the character and development of the eruption, the course of the fever, and the chronology of both. The papules were small and soft and pointed, and within twenty-four hours of their appearance a minute drop of pus had appeared at the apex. This was totally different from the round large hard deep papules of small-pox gradually developing into vesicles, and still more slowly into large multilocular pustules with a depressed centre; though some of those in our patient were distinctly

umbilicated, the great majority were not. The distribution also, though like that of variola, differed from it in the rapidity of its march. The pustules on the face, though closely set, in places did not coalesce, and those on the lower extremities appeared much too early.

The fever had preceded the eruption by three or four days, but it had not been more than 101°. It then continued to rise and it reached 103.6° on the evenings of the 28th, 29th and 30th of October; but it was not a continuous fever, it was decidedly remittent and irregular, much more so than enteric pyrexia, and resembling the course of septicæmia. On the whole, therefore, the evidence was conclusive against variola.

The alternatives were not many. Acne was disposed of, and varicella was negatived by the eruption not passing through a vesicular stage, by its abundance on the face and limbs compared with the back, and by its absence on the scalp. Moreover, chicken-pox is not common after puberty, and when it occurs is never accompanied by such a degree and duration of fever as in our patient. A pustular rash with remittent fever and pain and swelling of the joints may denote pyæmia, but in the present case there was no wound, no evidence of internal suppuration, and no endocarditis. Moreover, the absence of sweating, jaundice and prostration completed the negative evidence, and, after all, a pustular eruption in pyæmia is a rare occurrence and never, I think, so abundant as in this case.

One must never forget that anomalous rashes, papular, bullar, or pustular, sometimes accompanied with ulceration and granuloma, which looks like lupus or malignant disease, may all be caused by iodine of potassium in medicinal doses, and quickly disappear on its discontinuance. Here, however, the patient was taking no drugs and the pyrexia would still have needed explanation.

The diagnosis practically lay between variola and syphilis. Accordingly, after examining the eruption and the temperature chart, and inspecting the vaccination marks, one turned at once to the lymph-glands and found them hard, movable, smooth, painless, and swollen behind the sterno-mastoid, in the groins, and above the internal condyle of the humerus on one side. (Subsequently similar bullet glands were found above the left elbow and in each popliteal space.) We then examined the throat and the eyes, but found

neither iritis nor syphilitic angina. There was also no sarcocele. Besides these negative symptoms, the colour of the rash was remarkably free from the yellowish tinge which is known as "coppery." There was a moderate amount of irritation present, and the fever was unusual. Although the coppery colour, absence of itching, the presence of sore throat and iritis, and the multiformity of the cutaneous lesions are each and all valuable symptoms of syphilis, their absence did not materially weaken the evidence afforded by the other facts. We had in the inguinal scars the proof of a primary chancre. The pustular eruption answered to syphilis and to nothing else, and the enlarged glands were too small, too hard, and too painless to be the result of local inflammation. The pyrexia, though of unusual severity, was a symptom frequently observed and probably more frequently present in this disease; for syphilis is a specific fever, it invariably arises from contagion, it breeds true, it has its period of incubation, its exanthem, and its local lesions, and lastly it protects against itself. It would be strange then if it were unattended with pyrexia. A slight evening rise of temperature, with headache, and anorexia may often, perhaps usually, be discovered if looked for at the time secondary symptoms appear. Not unfrequently marked, and even severe fever occurs, the temperature rising to 103°, and even to 104°, and of this fact the present case is an example.*

This view of the pathology of Lues is really the revival of that which prevailed when it was first recognized in Europe. In the Latin poem of Fracastori, the title of which gave the name "Syphilis" to the disease, the hero, Syphilus, is a shepherd whose symptoms and their cure are described without any hint of an impure origin. The primary stage was not recognized for several years, and the disease was looked upon as an epidemic pestilence, infectious, no doubt, but only in the same way as the plague, the spotted fever, or the small-pox. It was really a retrograde step when, in the seventeenth and eighteenth centuries, the general infective fever syphilis was confounded with the local specific inflammation now known as soft sore, and with virulent contagious urethritis, under the common name of *Lues-venerea*.

You must learn to look at syphilis as a long-drawn-out specific fever, not always of venereal

* On referring to Mr. Hutchinson's " Manual on Syphilis " I find that he says (page 33), " The variola-like eruption in particular is almost always attended by much fever."

origin, to be recognized as we recognize small-pox or measles, by its exanthem and its other symptoms, and not by our being able in every case to trace its origin.

The diagnosis above stated was speedily confirmed by the pustules aborting far short of a variolous development, by fresh crops appearing, some so small as to be only just visible, others forming small bullæ containing thin pus, or large crusts with superficial ulceration. It thus became much more polymorphic or multiform, though the colour was never characteristic. Before long a lump appeared in one epididimis, the fever subsided, and the patient is now doing well under mercurial treatment.

Let me urge you to take every opportunity of seeing cutaneous rashes, if possible before a diagnosis is made. Make your own diagnosis and write it down; and, whether right or wrong, you will learn a valuable lesson. Remember that until your diagnosis is established, your treatment must be useless, if not injurious. If the present case had been variola, it would have been our duty to have notified it to the Medical Officer of Health, and to have had the patient removed to a small-pox hospital. You would also have done your best to prevent the disease spreading to others in the house, and you would give them the protection of a fresh vaccination. If you decided that the case was syphilis, you would have no such anxiety, and would only be concerned to get the patient as speedily as possible under the influence of mercury.

You are sure to meet with similar cases in practice, perhaps early in practice, and that will be an advantage, for you will not have forgotten the lessons of the wards, and will not be over-burdened with other duties. In such cases a correct diagnosis will not only be of the utmost consequence to your patient, but will affect your reputation and perhaps decisively. Do not, therefore, be in a hurry. Remember that diagnosis is often difficult, even to those most experienced. The man who decides hastily and dogmatically is sure to make disastrous mistakes—and deserves to. Go carefully over all the symptoms, and refuse to give an opinion until the facts have been ascertained and weighed. Do not be above confessing doubt, and on a second visit a few hours later, or on the following day, the case will have developed itself, and what was obscure will become clear. Always make a provisional diagnosis in your own note-book,

even if it be very general or an alternative one. In difficult cases seek the help of some neighbour whose judgment you respect; or, where you cannot get this, go through the case afresh on your second visit, in search of objections to your provisional diagnosis, and thus be your own consultant. A conclusion which withstands such criticism, and is strengthened by a second examination, is almost sure to be right.

ON THE OPERATIVE TREATMENT OF EARLY ACUTE APPENDICITIS.

By W. WATSON CHEYNE, F.R.C.S., F.R.S.,
Professor of Surgery in King's College.

AFTER reference to some cases, and points arising in connection with them, Mr. Cheyne proceeded as follows :—

It is only within comparatively recent years that one has heard of appendicitis at all. When I was a student twenty years ago, all these cases were spoken of as typhlitis and perityphlitis. Since that time, however, post-mortem examinations have shown the great frequency with which the appendix is the seat of perforation, ulceration, etc., and the pendulum has swung round in the opposite direction, so that it is now asserted by many surgeons that there is no such thing as typhlitis and perityphlitis, but that all cases of this kind originate in the appendix, and that therefore there is only one disease, appendicitis. I believe that in most cases the truth lies between these two extremes, and I would say that as a rule where a case begins acutely, and more especially where it ends in suppuration, it is one of appendicitis from the commencement, but that where the early symptoms are milder it may have originated either in the appendix or in the cæcum, and that even where it is ultimately found that the appendix is diseased it may have commenced in the cæcum and spread afterwards to the appendix. Where the cæcum is distended with fæces the inflammation of the mucous membrane may quite well spread to the appendix, and indeed if the distension is very great the valve may become stretched and actual particles of fæcal matter may pass through it. The existence of typhlitis as apart

from appendicitis has been confirmed by post-mortem examination, and possibly it is more frequent than is now supposed, because it generally ends in recovery, and indeed a good many of the cases of supposed appendicitis which recover without suppuration may be in reality cases of typhlitis. The symptoms that would lead one to diagnose typhlitis as opposed to appendicitis are previous constipation and dyspeptic troubles, gradual and not very violent onset of symptoms, pain in the iliac fossa, rather vague at first and gradually getting worse, loss of appetite, temporary obstruction, vomiting, fever, at first slight, and recovery in a few days. It is, however, where the case is more acute and lasts longer that the difficulty in diagnosis may come in. In typhlitis the pain is usually dull at first and subsequently increases. Pressure causes pain, even at an early stage, over a large area. The tenderness is limited to the region of the cæcum, and one is not likely to confound the case with general peritonitis. And, lastly, a lump can generally be felt at quite an early period, which is dull on percussion, and which in reality consists of fæcal accumulation. In appendicitis, on the other hand, the pain is from the first very severe, and reaches its maximum almost at once. The tenderness is at first at a limited point, about two inches inside the anterior superior spine of the ilium, and in a line between that point and the umbilicus. But very soon there is general tenderness over the whole peritoneum, sometimes to such an extent as to lead one to think that there is already a general peritonitis. And lastly, in appendicitis the swelling only appears some time after the commencement of the symptoms, and the cæcum is empty and resonant. While, however, maintaining the existence of a typhlitis, and even of a perityphlitis, as apart from appendicitis, I fully admit that by far the greater proportion of these cases commence in the appendix, and from a practical point of view appendicitis is of much greater importance than typhlitis, and I may therefore say a few words as to the forms and general symptoms of appendicitis.

There are two chief forms of appendicitis, viz., with and without perforation; and the cases with perforation may again be divided into those with acute general peritonitis and those with localized peritonitis.

Acute general peritonitis in a child is, in the absence of any other evident source, practically always due to perforation of the appendix. It

commences with sudden violent pain in the iliac fossa, or near the umbilicus, sickness, and perhaps vomiting, rapid generalization of the pain over the abdomen, fever, and all the other symptoms of peritonitis, into which I need not enter. As a rule, the tenderness is greatest in the region of the appendix, and there also distinct fulness and hardness may be felt, especially if the patient is placed under an anæsthetic ; and fulness, especially in the right iliac region, may be made out *per rectum*. This is a very fatal form, and the treatment must be particularly energetic. As soon as signs indicating extension of peritonitis over the abdomen have arisen, in cases where there is no other evident cause, no time should be lost in performing laparotomy, and as by far the great majority of these cases are due to perforation of the appendix, the incision should in the first instance be made over it, *i.e.*, about two inches internal to the anterior superior spine, the centre of the incision corresponding to a line drawn from the umbilicus to the anterior spine. I believe this is the best practice, whether fulness be felt in the iliac fossa or not, because it is useless in such cases to wash out the abdomen and leave the original focus behind ; and besides, the washing is carried out better through two holes than through one. Having opened the peritoneal cavity, the appendix should be at once searched for and removed, and in these cases there is no objection to doing this, because one does not make the communication with the abdominal cavity worse. The next step is to flush out the peritoneal cavity, but before doing so it is well to remember that, although the symptoms may point to general peritonitis, the suppurative infection may, after all, not be general, and therefore, having thoroughly disinfected hands, etc., again, I make a small incision in the middle line in order to see if there is general peritonitis ; if not, I content myself with draining the original opening, and stitch up the central one. I have had more than one case of this kind which recovered. If there is general suppurative peritonitis, then this incision can be enlarged, the adherent coils separated, and the cavity flushed out as thoroughly as possible with boiled saline solution.

Probably in the majority of cases, however, the actual perforation has been preceded by a localized peritonitis, so that when the perforation does take place the appendix is already shut off to a certain extent from the general peritoneal cavity, and may

remain so. In other cases, even in the previous absence of adhesions, they form quickly and shut off the cavity. In other instances, after a preliminary period of diarrhœa and indefinite illness, symptoms not so acute as the above, but similar, appear, and then after three or four days things quiet down and the inflammation is found to be localized. In these cases there would not be any questions as to the urgency of operative interference or as to the procedures to be employed.

I shall therefore pass on to the much more difficult set of cases where the symptoms are not so urgent and where in all probability there is no perforation in the first instance, if at all. Even here the first symptoms are generally acute, violent paroxysmal pain beginning in the iliac fossa and radiating over the abdomen, vomiting, and the temperature rising gradually. Pressure causes pain over the appendix. After three or four days these symptoms in mild cases may improve, induration can be made out with a clear note over the cæcum and colon, and the attack passes off.

In other instances, however, improvement does not occur so early, and it is these cases which are so difficult as to the question of suppuration or as to the question of operation. Of course, if one does not see the case until there is a large abscess, there can hardly be any doubt, the temperature will be of a hectic type, there will be a swelling of considerable size, a history of continuance of the symptoms, increase in the swelling, and possibly the occurrence of rigors. Here there cannot be any difficulty, but as in cases left so long the patient has run great risks, it is, as a rule, necessary to make up one's mind at a much earlier period as to the presence of pus and as to the advisability of operation. One very important point to guide us is the length of time that the symptoms last. We know that in the case of a very acute inflammation of the subcutaneous tissues, suppuration will have occurred in children in two or three days, and in adults in four or five, and probably the time is about the same in the case of the peritoneum. In any case I think we shall very seldom be wrong in assuming that if an appendicitis has lasted without remission of symptoms for five days at the most we are pretty certain to find pus. I know that cases have occurred where the acute symptoms have lasted longer and recovered without operation, that is to say, without the formation of pus, for I do not believe that in such cases pus would either become absorbed or remain quiescent. Such

cases are, however, quite exceptional, and we should lose many more patients than we do if we took them as our guide. Further, the occurrence of one or more rigors adds to the certainty of suppuration; at a later stage also there is œd-ma of the skin. A hectic type of temperature, wasting, sweating, increase in size of the swelling, in spite of free evacuation of the bowels, are also of importance. It is under these circumstances that the question of treatment presents the greatest difficulty. As the question which interests me most, and which is, after all, of the greatest importance, is the surgical one, I shall not enter in any detail into the purely medical treatment. Rest to the bowels, fomentations or poultices, opium and fluid diet are the methods usually employed, but there is one other thing to which I would call attention, and that is the value of the so-called intestinal antiseptics, more especially of naphthalin. I believe that in many cases the trouble is largely dependent on or, at any rate, aggravated by decomposition occurring in the cæcum and the retention there of these decomposing materials. As a rule, one dare not give a purge to clear out this material, and therefore the best thing is to try and diminish it as far as possible. I must confess that theoretically I am doubtful as to the efficacy of intestinal disinfection, but practically I have seen great benefit from it. I have used naphthalin in several cases where the question of operation has had to be considered, and benefit seems to have followed its use.

The question of vital importance then, which has to be faced as soon as the diagnosis of appendi-i is has been made, is that of operation. As I have already said, where the symptoms are very acute and the peritonitis is spreading, there can be no difficulty, but in the cases which I have just been referring to where they are not so acute, and the peritonitis remains localized, one is often very puzzled as to the right thing to do. However, once pus has formed it ought to be evacuated without delay, and I have already indicated some of the points which lead one to suspect the occurrence of suppuration. As a matter of fact, if the acute symptoms have lasted more than four or five days it is best to operate whether one is sure of the presence of pus or not. Puncture to ascertain the presence of pus need only be mentioned to be condemned. The conclusion I have come to is in favour of fairly early operation, operation whenever there is considerable reason for assuming that pus

has formed whether one is sure of it or not. I do not, of course, mean that every case in which acute symptoms last for more than five or six days should therefore be operated on; one must take into account all the circumstances of the case, and I only suggest this time as a sort of guide, and I think the same rule holds good here as in the case of hernia, viz., when in doubt, operate.

Suppose no pus is found, what are the disadvantages? In the first place, it is supposed to be dangerous to open an inflamed peritoneum, and hence in such cases as ovarian cysts, with active peritonitis, operation is deferred till the inflammation subsides. This is a relic of the experience of the old septic surgery. It is, no doubt, true that where no antiseptic precautions are taken, an inflamed peritoneum, having lost its protective power, is much more likely to suppurate than a healthy peritoneum, but if the operation is performed strictly antiseptically there is no such risk; on the contrary, we know that incisions into inflamed parts do much good, and this is also the case in peritonitis. A real disadvantage, however, both of early and late operation, is the weakening of the abdominal wall, which is apt to result. This can only be guarded against by taking care to stitch up the abdominal walls properly, more especially by making valvular incisions, stitching the various layers separately, and more particularly employing Macriven's method of catching hold of the muscular planes. This is, to my mind, the essential obstacle to early operation—viz., that one does not wish to leave the patient with a weak abdominal wall. Another thing which has been mentioned as a disadvantage is that, if pus is present, and one operates too early, one may not find the intestine adherent to the abdominal wall, and thus the general peritoneal cavity may be soiled. There is, no doubt, some truth in this objection, but my experience is that this fear of soiling the peritoneal cavity is much exaggerated, and that if the region in which the pus was present is only well drained there is not much to fear. I have seen this in several cases, but perhaps the most remarkable as showing how free drainage can obviate the risk of infection of the peritoneal cavity is one, not of appendicitis, but still sufficiently interesting to mention. It was that of a young lady who, eighteen months before, had been under the care of eminent gynæcologists and ovariotomists for what was supposed to be suppuration in connection with an ovarian cyst. The

pus had opened into the rectum, and an opening was subsequently made above the pubes and a drainage tube put into the cavity. The suppuration went on, large quantities of pus being discharged into the rectum and also through the front wound, the patient was rapidly getting weaker, with hectic temperature, night sweats, etc., and those in attendance did not feel inclined to do anything more. I saw the patient, and decided to give her the chance, as she was evidently dying rapidly. I therefore opened the abdomen and removed the mass, which proved to be tubercular, and involved the right ovary and Fallopian tube, and adhered to bladder, uterus, and rectum. The peritoneal cavity was healthy, and the result of the operation was that a hole was left in the rectum communicating freely with the healthy peritoneal cavity, and the intestines had, no doubt, been partly soiled during the removal of the mass. I put a number of drainage tubes (five) through the anterior opening right down to and around the hole in the rectum, and that patient never had any general peritonitis, and recovered.

In spite of these objections, early operation has advantages which I think fully counterbalance the disadvantages. In the first place, as I have already mentioned, the opening up of inflamed tissues is one of the best curative measures, and in the second place, if no pus is found there can be no possible objection to removal of the appendix, and by doing so the whole trouble is cut short, and much present and future risk is avoided. Naturally, in such a case a drainage tube would be kept in for some days. Even if pus is found, and the part is not shut off from the general peritoneal cavity by adhesions, I do not think there is any special risk. Sponge out this pus and put in drainage tubes, and the chances of extension are slight, not so much, I think, as if it is left.

One other point with regard to operation during the first attack of appendicitis, and it is this. If we cut down and find pus are we to content ourselves with simply opening the abscess, or are we to search for and remove the appendix? The usual procedure is to be content to open the abscess and not to look for the appendix for fear of soiling the peritoneum. I quite agree that where the abscess is large, or where, as too often is the case, the surgeon is not called in till very late, and the patient is too ill to permit any prolongation of the operation, one should be content with opening the abscess, but where there is only a little pus,

and the patient's condition is good, I think it is best to search carefully for the appendix, and if possible remove it. If it is left the patient is constantly liable to recurrences, and often the wounds are long in healing, and I am satisfied that the fear of soiling the peritoneum is a much exaggerated bugbear. Where the quantity of pus is small it can be all sponged up, and after removal of the appendix a couple of drainage tubes should be passed down to the part.

A CLINICAL LECTURE
ON
PUERPERAL AND LACTATIONAL INSANITY.

R. PERCY SMITH, M.D., F.R.C.P.,

Resident Physician to the Hospital.

GENTLEMEN,—We are going to-day to consider, in the first place, cases of insanity occurring in connection with childbirth, which are among the most interesting, and, fortunately, among the most curable cases to be met with in asylums. As we have not at present any patient suffering from the more acute form of puerperal insanity in the hospital, it will be well to narrate at first the case of a lady who was recently here, and who has been since discharged on recovery. This patient, aged 28, was admitted here June 9th, 1893, suffering from acute puerperal insanity. The family history was that her father had been for twelve years, and was still at the time of her admission, confined in an asylum, his insanity being the effect of alcohol and business worries, and that a brother had also been alcoholic, and had committed suicide. In her personal history the only noteworthy facts were that she had always been of a nervous disposition, that menstruation had generally been painful though regular, and that she had had two severe attacks of influenza, three years and eighteen months ago respectively. There had been no special trouble during pregnancy. She was delivered of her first child (a male) at the end of May, 1893, the labour was perfectly natural, and there was no complication. Five days after confinement, however, she became somewhat de-

pressed, and took a dislike to her nurse ; maniacal excitement rapidly supervened; her cheeks became flushed, her eyes bright and staring, and she was frequently shrieking and singing. She had hallucinations of sight and hearing, refused all food, and constantly tried to get out of her room ; she recognized no one, was very violent, and her speech was incoherent. She threatened to destroy herself, her husband, and child ; attacked her nurse, and attempted to gouge out her eyes. She was admitted here six days after the onset of these symptoms. On admission her tongue was foul and dry, her lips and teeth were covered with sordes, and her temperature was 100 6°. She was drowsy, and her pupils were widely dilated, probably as a result of the administration of hyoscine before admission. On recovering from the effects of the drug she became very excited, constantly tossing herself about on the floor of the room in which she was placed, and muttering incoherently or calling out at intervals. She could give no intelligent replies to questions, resisted violently any attempt to do anything for her, and refused all food. There was a normal lochial discharge, and there was no evidence that the pelvic organs were in any than the usual condition after childbirth.

In consequence of her absolute refusal of food it was necessary to feed with fluid nourishment by the nasal tube, and paraldehyde was given to procure sleep. A few days after admission she was somewhat improved, showed less resistance to attention or feeding, the temperature became normal, and the lochial discharge ceased. It was now very evident that she had auditory hallucinations. By the 19th of June (ten days after admission) she was taking food without artificial aid, but yet continued to mutter to herself, or to imaginary persons, and was at times very excited and with difficulty prevented from getting out of bed, in which it was thought well she should remain for the sake of rest, in order to avoid, if possible, further waste of strength. The outbursts of maniacal excitement gradually disappeared, but were replaced by great mental depression, with the delusion that accusations had been made against her character. Hallucinations of hearing continued, evidently causing her great distress. Her physical health now improved greatly ; and her sleep became more natural, although she frequently needed medicine to produce it, chloralamide now being used in doses of grs. xl, subsequently reduced to grs. xx. At the end of July she still

had hallucinations, thought she was the devil, was dirty in her habits, and became very suicidal, attempting to choke herself, to dash her head against the wall, and precipitate herself from any height she could get to, and could not be left alone for a moment. She had, however, lost all homicidal feelings, and never in any way interfered with anyone. By August 21st she was much quieter, and talked rationally for the first time, she realised that she was in a hospital, but had lost count of time, and knew that she must have been ill, as there was this gap in her memory. This improvement, however, was only temporary, and was followed by a relapse into a condition of depression again, and then by a condition of semi-stupor from which she could be roused to answer questions in a whisper. In this state she did not seem to have any hallucinations or delusions, although she sat alone avoiding other people. Her physical health still continued to improve, and she began to get quite fat. This condition of semi-stupor and apathy, however, lasted from August till February of this year, and then gradually there was an improvement. She began again to employ herself with needlework, and to talk more to other people, and to take some interest in her surroundings. All this time menstruation had not been re-established. Toward the end of February she had so much improved, that it was possible to send her to our convalescent home at Witley, where she remained six weeks, and there still further improvement took place. She gradually became brighter and talked naturally. She had lost all delusions, and fully realised that she had been ill. She looked healthier, and the last evidence of restoration to health was the restoration of the menstrual function. In April of this year she returned from the convalescent home, and was discharged as recovered, her whole illness having lasted ten months.

This case illustrates many of the most familiar features of puerperal insanity. In the first place, here is a woman of nervous temperament and nervous inheritance who has her first child (a male), and breaks down under the combined effects of shock to the nervous system produced by the act of parturition, and the effect of the consequent changes in circulation and nutrition which follow childbirth. Even to those in whom the nervous system is healthy both by inheritance and development a first confinement must always be a severe strain, although the effects differ, of course, enormously in individuals ; but to those

belonging to neurotic or insane stock, or who are of nervous temperament, the danger of the nervous system giving way at this period is infinitely greater. Dr. Clouston found insane inheritance in nearly half the cases in which the family history was inquired into. I have not been able to find quite the same frequency here, but of 100 consecutive cases which have been admitted to this hospital in the ten years 1885–1894 inclusive, I have only been able to find a family history of insanity in 25, although this point has been always carefully inquired into. In some cases there seems to be the direct inheritance of a tendency to break down after parturition, the patient's mother having previously suffered from puerperal insanity. In one case admitted here in 1892 both the mother and daughter had each broken down for the first time at the birth of a fourth child, and in neither case did recovery take place. There seemed to be an exact parallelism. The next point is that it is at the birth of a first child, especially if it be a male, that insanity is most likely to result. Of the 100 cases mentioned 41 were first confinements. Then the other cases are as follows :—

At the birth of the second child, 14; at the birth of the third, 16; at the birth of the fourth, 10; at the birth of the fifth, 8; at the birth of the sixth, 5; at the birth of the seventh, 3. In two cases insanity followed the tenth confinement, and in one case the twelfth. When puerperal insanity occurs after the birth of a child born subsequently to the first three, it will commonly be found either that there has been a previous puerperal attack or some other attack of insanity, or that there has been a serious moral strain of some kind, or some special complication which has over-weighted the nervous system at a time when quietude is specially necessary. This seems to have occurred in all the above cases where there were more than three children. For instance, the patient who was admitted here after the birth of her twelfth child had had two previous puerperal attacks. In a very large proportion of these cases the labour seems to be uncomplicated, as in the case narrated, but in 43 of the 100 cases there had been either the use of forceps and anæsthetics, or the latter alone, or ruptured perineum, post-partum hæmorrhage, or some pelvic inflammation or septic trouble.

A patient was admitted here in May of this year, 27 years of age, having had her first child three months before admission. There was no insane inheritance, and the labour was easy and normal. There was no use of chloroform or forceps, but two days after confinement "puerperal fever" was diagnosed by her medical attendant. In spite of careful treatment she became extremely ill, had what was called an "abscess in the side," which one supposes was pelvic. She was sent to Lambeth Infirmary, where she remained extremely ill for some few weeks, and then she was transferred here on the 25th of May, the illness having lasted eight weeks before admission. She was excited, her speech was incoherent, she raved and shouted, was destructive, and also dirty in her habits. But in spite of all this, and in spite of the septic trouble, her strength was maintained. When admitted here, although she was very thin—the effect of the excitement—her appetite was good, and she was able to take abundant nourishment, and very soon there was a very great improvement in her physical health. There was then no evidence of any septic trouble; her temperature was normal, and there was no reason to think that there was any special pelvic disease. Improvement rapidly took place, and she was discharged from this hospital as cured about three months after admission. The reason I mention that case is because she was a patient in whom there was certainly some septic trouble at first, in spite of which, however, she recovered fully. On one occasion when, soon after her admission, Dr. Rohé, of the Maryland Hospital, Virginia, came round the hospital with me, he expressed the opinion that the excitement depended on some permanent uterine or pelvic irritation, and that the proper treatment would be to have the uterine appendages removed to promote speedy recovery. With regard to the removal of the appendages in such cases, it is not a practice which is adopted in this country; and I think the fact that the patient recovered perfectly and remained without any symptoms of any uterine or ovarian disease, proved that there was no necessity for such an operation.

With regard to moral causes, it is well known that mothers who give birth to illegitimate children are specially liable to puerperal insanity. One has not happened to meet with such cases in this hospital, but in Dr. Clouston's cases at Morningside no less than 25 per cent. of the puerperal cases followed illegitimate birth. Of course, at Morningside patients of all classes are admitted, whereas here we only receive people of

the educated classes, and that may perhaps explain to some extent the difference. I have, however, frequently seen cases in which there has been some other moral shock or emotional strain, and especially cases in which the death of a near relative or of the husband about the time of delivery, or just after delivery, has seemed to be the "last straw" which has caused the patient to break down. In one case, fright from seeing an insane woman at the time, and in another case, the exciting cause was undoubtedly an attack on the house by "moonlighters" (this was in Ireland, the patient being subsequently admitted here); so that any nervous shock coming just after a woman has been confined, may precipitate an attack of puerperal insanity. Then in several cases nervous apprehension of the pains and dangers of delivery has been present, and this generally in primiparæ of advancing age.

As in the case quoted above, the attack, if one of maniacal excitement, usually commences within the first fortnight after delivery, the cases characterised by depression usually beginning later. Of the 100 cases mentioned above, 57 began within a fortnight of delivery, and the large majority of these were cases of acute mania. In most cases there is a period of depression, with sleeplessness, but sometimes this is exceedingly short, and in one case admitted here the patient is said to have "woke up raving." She had apparently been going on perfectly well before, and had gone to sleep, so that there was no warning of the attack. Perhaps, however, the friends did not carefully observe any depression or alteration of demeanour. But whether there is a short depression or not, in most cases the attack develops with alarming rapidity, and dangerous symptoms soon appear. These very commonly consist of attempts to injure self or husband or child, and, as in our case, there may be suicidal attempts, even though the patient is not at first melancholic. In a very large proportion of cases there is a dangerous explosiveness, ending in some serious act. The special danger to the child must, of course, never be overlooked. This may arise simply from neglect, the mother not being able in her confused state to take care of her child or to perform the usual maternal duties, or the child may not be recognized as such, and may be thrown down on the floor just as a pillow or a bundle of clothes might be; or, if the mother is foolishly allowed to take care of her child, there

may be some unreasoning act, such as holding it close to a fire, or putting it too long in the water; or there may be hallucinations which cause the mother to destroy her child. She may hear a voice telling her to do so, or she may have some delusion about it—that it is black, or deformed in some way—and may therefore kill it. In some cases a sudden frenzy leads to its unreasoning and almost unconscious destruction. A good many cases of puerperal insanity find their way to Broadmoor Asylum as criminal lunatics. People who have suffered from puerperal insanity, and who have not been properly watched, have suddenly killed their children, been tried for homicide, and sent to Broadmoor, where they may recover from their puerperal insanity; but, of course, for the protection of the State it is usually necessary that they should remain a long time under care.

A good many cases are admitted here every year, where, fortunately for the mother, there has not been such a serious result as that, but where there has been an attempt to injure the child. I am frequently able to point out cases admitted here where the mother has only just escaped becoming a "criminal lunatic" by timely intervention. Next to the special dangers of suicide or homicide there comes the danger to the mother's life from the nature of her physical and mental illness. Although there may be no serious local complication there is often some offensiveness about the lochial discharge, some tenderness about the uterus, and perhaps some slight temporary rise of temperature. These symptoms, of course, need very careful attention, and the local conditions require proper treatment. But in a very large proportion of cases, as in the one I first narrated, there is no evidence of any local pelvic disease, nor is any uterine medication needed. The dangers do not lie so much in this direction as in the exhaustion which may rapidly ensue as the result of prolonged and intense maniacal excitement, with sleeplessness and refusal of food. In these cases the importance of getting the patient to take abundant nourishment, if not voluntarily then compulsorily, by artificial means, cannot be too much insisted on. She must be fed, if necessary, three or four times a day, by the nose or œsophageal tube, with milk, eggs, beef tea, or meat extracts, and, if the pulse seems weak, with stimulants, and there must be no cessation of this till she is able to take sufficient food, not merely to sustain life, but to cause her weight to increase.

A very large proportion of these cases of acute mania need stimulants, the pulse being feeble and rapid. Sometimes stimulants have been countermanded by the medical man in charge of the case before admission on the ground that the condition is one of " brain fever," and that if stimulants are given the condition is made worse. That is a mistake, as one finds that a very large proportion of these cases need stimulants. Next to feeding, the procuring of sleep must be attended to, and here we generally give either paraldehyde, sulphonal, trional, or chloralamide in acute cases. Morphia does harm, and chloral interferes with the heart, and is apt to make the patient anæmic, —as a matter of fact, it is hardly ever given.

In the great majority of cases the only proper course is to have the patient promptly removed to an asylum. In most cases she is quite unmanageable and unbearable in an ordinary house, and the curability seems to be directly in proportion to the shortness of the period allowed to elapse between the onset and removal to an asylum. Then with regard to the character of the patient's insanity, in these cases of puerperal maniacal excitement one does not, I think, as a rule see very much to distinguish it from other cases of acute maniacal excitement. Bevan Lewis lays considerable stress on the presence of eroticism as a special symptom in these cases, but I cannot say that my experience has been the same here. Of course in some there is marked eroticism, but so there is also in many cases of acute mania in young women. In the case I have mentioned there was no eroticism. It always seems to me that there are numbers of cases of acute puerperal mania, in which, if you were not told that the case was a puerperal one, you would not necessarily be able to infer it from simply seeing the patient.

As in the case narrated, excitement may be followed by profound depression, or a condition of temporary stupor, in which the nervous system, or at least the higher intellectual sphere, seems torpid, the appearance suggesting at the time permanent impairment of mind. The condition, however, is commonly only a temporary one, and with persistent feeding and care recovery follows. This condition is almost invariably associated with amenorrhœa, which, however, need give no special anxiety, as in the large majority of cases menstruation returns with improved general health, the nervous system seeming to right itself at the same time. It is no bad guide to say that in these cases the prognosis is good as long as there is amenorrhœa, but that if menstruation returns and becomes regular without a simultaneous mental improvement, the likelihood of recovery is much lessened. Puerperal insanity is one of the most curable diseases if taken in hand early and energetically. Of the above-mentioned 100 cases, 75 recovered and only two died ; five others were discharged as relieved, being able to return home, although not perfectly recovered mentally. That leaves 18 in which the patients remained uncured. Of the cases that remained uncured, the average duration of the attack on admission was three months, showing, as I said before, that the curability seems to be in proportion to the earliness with which the patient is admitted to hospital treatment. It should be stated that patients do not, as a rule, remain longer than twelve months in this hospital if they have not recovered, but are removed elsewhere, and therefore some of the 18 cases discharged " uncured " may have subsequently recovered.

As an example of puerperal melancholia, one may mention the case of a woman who was admitted here a couple of years ago, suffering from depression which supervened after the birth of her first child. She was 39 years of age when the child was born ; the labour had been tedious but not instrumental, and she had subsequently remained weak and exhausted. The attempt to nurse the child seemed further to exhaust her. Between one and two months after confinement she began to be depressed, accused herself of imaginary crimes, and thought everybody was against her. She was also very suicidal and made attempts to injure herself, although she did not attempt to injure her child. Treatment at home was tried for a time ; in fact, it had been tried for no less than eight months before she was admitted here. Fortunately, she began to improve, and removal from home surroundings, careful feeding, and attention to sleep, etc., ended in her recovery; but possibly if she had been admitted sooner than that, her illness would have been of much shorter duration.

In a few cases the form of insanity is neither maniacal nor melancholic, but the patient becomes weak-minded from the first, and never seems to improve ; and in some other cases chronic delusional insanity with ideas of persecution sets in, and the patient has to remain in an asylum for the

rest of her life. In one of the above-mentioned 100 cases, general paralysis appeared to start immediately after delivery, but this may have been a coincidence. The friends may not have carefully observed any alterations in the patient's mental condition before, and of course one knows general paralysis generally has a premonitory period in which there is some alteration, but the friends dated the attack from delivery.

The patient whom I now bring before you is an instance of chronic delusional insanity dating from confinement. Mrs. P., 40 years of age, who has four children living (the youngest having died five months after birth, and only two months before admission here), was admitted on February 2nd, 1894. Her father was what was called "nervous," and all the female members of the family were hysterical, and one brother was alcoholic, but there was no evidence of definite insanity in the family. She has had two previous attacks, the first in 1879 (a puerperal one), and the second after a miscarriage in 1892. She was confined in July, 1893, and within two months she had slowly developed delusions of persecution. She suffered, and still does suffer from hallucinations of all her senses, hearing voices which give her commands through tubes, seeing imaginary faces, noticing bad tastes and smells, which she thinks are due to poisonous materials that are sent to annoy her, and feeling twitchings, or pinchings, which she thinks, are due to electricity, which is conveyed to her by various machines. She believes that all this is the work of enemies who wish to make her life a burden, and she is constantly demanding police protection from these annoyances. One may say that considering the duration of the attack, the fact of previous attacks, and the character of the mental disease, the prognosis is extremely bad.[*]

Prognosis.—The majority recover, but one has to remember that there is a liability to further attacks, especially after confinement. One occasionally sees patients here who have come with perhaps their fourth or fifth puerperal attack, having had an attack after the birth of each child. One dreads to think about what is likely to be the future of all those children. They are sure to be extremely unstable, and very likely to break down in adolescence; if this does not occur they are, if females, exceedingly likely to break down at the puerperal period. Occasionally one sees women who have had a puerperal attack after the birth of the first child bear several children without breaking down again, but subsequently becoming insane at the climacteric.

With regard to those who seem to acquire a sort of habit of puerperal insanity it is most important that they should not continue to have children; in fact one would be inclined to say that when a woman has once had a puerperal attack it certainly is best from all points of view that childbearing should cease, and it is imperative to impress this fact upon the husband and wife.

With regard to lactational cases, the border-line between puerperal and lactational insanity is rather an artificial one, and is drawn rather arbitrarily by some at six weeks after confinement and by others at two months after, but it is very difficult, for instance, in a case beginning three months after confinement, and where the woman has nursed her child, to say that the origin of the attack has not been connected with the changes immediately following parturition. In the same period covered by the 100 cases above mentioned, there have been admitted here 35 cases of lactational insanity. Of these 14 have been maniacal and 18 melancholic, the proportion of the latter form being therefore greater than in puerperal cases. In this condition, instead of the severe shock of parturition, which seems so frequently to rapidly disorganise and to profoundly upset the nervous system, there is, on the contrary, generally a slow drain on the energies, which, in most cases where there has been very prolonged suckling, has led to general debility and anæmia; here the melancholic form of mental disease seems to predominate. But at the same time I ought to say that of the above cases the one where there had been the longest period of suckling (20 months) suffered from acute mania, which came on rapidly five days before admission. Nearly all these lactational cases are anæmic; in all the general health is weak and the nutrition poor, and all need abundant food, sleep, and tonics. Of the 35 cases 27 (or 77 per cent.) recovered, and therefore the prognosis may be said to be favourable as a rule, and there is certainly less danger to life in acute lactational than in acute puerperal insanity. Of course, there is the absence of the possibility of septic complications.

The following case will illustrate insanity coming on during lactation: The patient, who is 33 years of age, was admitted on the 31st March,

[*] Dr. Smith here showed one or two instances of slow convalescence with amenorrhœa.

1894, and she has two children, the youngest of whom was three months old on admission. She had nursed it until that time. There was no family history of insanity, and she had had no previous attack. About three weeks before admission she had become sleepless, had taken a dislike to her child, and had heard a voice saying " Won't you kill the baby and your husband?" and also suggesting immorality. She began to think that some unseen agencies were working upon her, and that they had tampered with her in various ways, and dipped her hands in vitriol ; she also thought that some of her organs had been removed. On admission she was extremely pale, very anæmic, restless and confused. She believed she had been hypnotised and "influenced" and that she had two or three identities. She thought she saw a doctor come out of a chimney, and she also noticed the smell of sulphur, and thought she was shrivelled up. There were hallucinations of various senses very much like the puerperal case we saw. These apparently grew rather gradually, and it was hoped that with an improvement in the general health the symptoms would slowly disappear. I am afraid, however, that the prognosis in that case is not very favourable. She has been here since March. Of course, that is not too long for recovery, but she has many sensory disturbances still, and I am afraid she is becoming more and more convinced that she has been persecuted and tampered with in various ways, and that there will always be permanent delusional insanity resulting. Menstruation has returned, but yet not very regularly ; her general health has improved, she has more colour, and is not flabby and weak as she was, but unfortunately there is no corresponding mental improvement. Therefore one is afraid she is not likely to do well.

The next patient is also suffering from lactational insanity. This is a much more acute condition, and, I think, more likely to be cured. The patient, who is 34 years of age, was admitted here on October 5th last. There is no inheritance of mental disease. She had one previous attack two years ago which only lasted a few days after nursing for five months. She had a child on February 22nd of this year, the labour being long and difficult. An anæsthetic was administered. She nursed the child till September 9th, and then there was some kind of febrile attack, whether influenza or not I do not know. She then ceased nursing, and depression came on at the same time ;

there were suspicions and impressions that. everybody was against her, and she became very agitated, refused food, was very restless and resistive, and had to be admitted here. On admission there was the same condition—depression with agitation, constantly hearing her children's voices, needing to be washed and dressed and fed and looked after in every way. She is certainly less anæmic than she was when she first came in ; her appearance is more healthy, but there is always restlessness and inability to converse or give her attention. There is no doubt that she has hallucinations of hearing, and there is still resistance to dressing, to washing, and to food, but I think she has slightly gained flesh. She is worried about something which she cannot quite formulate, and she is confused and out of health. It is a case where one can say that the patient is so much out of health that there is considerable margin to make up, but there is every reason to expect that with good feeding, iron, and sleep, she will steadily improve.

CLINICAL DEMONSTRATION OF CASES.

Two Cases of Pigmentation.

DR. KNOWSLEY SIBLEY exhibited two cases of rather unusual pigmentation, one in a woman of 42, and the other in a young woman of 20.

1. J. B., aged 42, a widow, the mother of eight children. She had always been healthy until about six years ago, when she had ovaritis ; and about one year and a half ago she suffered from peritonitis. For the past three years she had been subject to loss of strength, general nervousness, and palpitation ; and three years ago her friends began to notice blood spots appearing on her lips. She had a considerable number of pigmentation spots on the lips and hard palate, the amount apparently increasing. When under examination her pulse was generally about 140 ; but when not noticed quieted down to a little over 100. No distinct cardiac disease, but the second aortic sound somewhat accentuated, and occasional mitral systolic murmur. DR. SIBLEY believed the pigmentation had to do with pelvic conditions, and the case was analogous to some of those DR. CHAMPNEYS had recorded in the St Bartholomew's Hospital Reports a few years ago. The patient improved under ordinary treatment to a certain extent. There was no pigmentation in other parts of the body.

2. P. R., aged 20, single; with pigmentation on face, axillæ, hands, and areolæ, the latter pigmentation being of a remarkable character. She first came to DR. SIBLEY under the mistaken impression that she had jaundice. The conjunctivæ were perfectly normal. She suffered from nervousness and prostration very similar to the symptoms in Case I, and also had a very rapid pulse. There were one or two spots in the mouth, on the buccal membrane. She also suffered from dyspepsia. The catamenia had been absent for about three months. In DR. SIBLEY's opinion it was a case of gestation.

DR. CAMPBELL said that the most interesting feature of the cases appeared to be the liability to attacks of prostration. Although most people would regard the second case as one of simple pigmentation, he was rather inclined to view it as a case of Addison's disease.

DR. GILL asked DR. SIBLEY his opinion as to the cause of the hæmorrhage about the lips and the hard palate in Case I, and whether it was due to any general disease. He had seen similar pigmentation in scurvy.

DR. GUTHRIE said he was inclined to regard the cases as Graves' disease. The acceleration of pulse, palpitation, and attacks of prostration were all in keeping with Graves' disease, and it was by no means uncommon to find pigmentation in that condition equal to that of the younger patients.

DR. MILSON asked DR. SIBLEY whether he had noticed instances of pigmentation in people who had lived in malarial districts, and who were subject to ague.

DR. SIBLEY, in reply, said he had never entertained the view that the cases were scurvy. Undoubtedly the pigmentations were hæmorrhage originally, but they were not altogether so at the present time. Putting all the facts together he thought DR. GUTHRIE's suggestion that they were Graves' disease was nearer the mark. He had noticed that although people suffering from living in malarial districts do get pigmentation, they also get it in a number of other diseases. He believed pigmentation was certainly a nervous condition.

Acromegaly.

DR. HARRY CAMPBELL exhibited a case of acromegaly, which he considered the best case living. The patient, a male, 48, presented all the classical signs of the disease. The voice was deep and hoarse, the legs bowed, and there was a remarkable development of the pelvis on either side. There was a decided transverse occipital crest, which, as they all knew, was very greatly developed in the anthropoids, and the patient also had prominent supra-orbital ridges, prominent eyes, and large jaws. The lower jaw was especially large. The septum and fleshy parts of the nose were greatly hypertrophied, and the nasal bones were elongated. There was an anterior projection of the upper jaw fully three-quarters of an inch from the rest of the jaw. The neck was thick, and the chest considerably enlarged antero-posteriorly. The hands were unusually large. They were comparable to an infant's hand exaggerated. There was also a remarkable development of the tensoris vaginal femoris muscles, and this he found was a feature among the larger apes. DR. CAMPBELL pointed out in detail the similarity of certain characteristics exhibited by the patient to those exhibited by the anthropoids, and suggested that the patient possessed many of the characteristics of man's ape-like ancestors.

The patient himself said, "I am an instance of a human being without the form of a human being, growing backwards."

DR. ABRAHAM said he did not think the patient showed many points of anthropoid character. He believed the patient's bones had been growing for the last fifteen or sixteen years. The muscular development was not in proportion to the skeleton. Another point was that the disease did not come on until adult life.

MR. BRODIE said that although the patient's arms did not reach below the knee, as in the gorilla, there was the same sort of attitude.

DR. CAMPBELL acknowledged that there were many arguments against the idea that the case was one of "reversion," but he did not think DR. ABRAHAM had advanced anything against his theory, because it was quite possible for a fully developed individual to grow backwards. That was a principle not properly recognised in disease. His belief was that "reversions" were displayed in all diseases.

Heart Disease.

DR. CAMPBELL also brought forward a case of dilated heart in a woman of about 50 years of age. He said he had treated her upon the orthodox lines. She was put to bed, given digitalis and so forth, and the result was that she got no better. Her pulse was about 160, and evidently the heart was only pumping at each beat a small quantity of blood. She was water-logged, and in a very serious condition. He then began to treat the case on what he would call "rational lines." The patient was stout, and, as it seemed to him the right thing to do was to get rid of the fat, she was put on milk diet. In order to avoid gout he gave her some salicylate of soda. Under that treatment the fat was reduced. In all cases of chronic heart disease, where the patient was stout, the first thing was to get rid of the fat. As soon as the patient had got rid of the fat there was less work for the heart to do; and the diaphragm, instead of being pushed upwards, was able to descend. Breathing became freer, and the circulation improved. The result of the very simple treatment of getting rid of the fat, of getting the patient to take frequent deep breaths, and of putting her through one or two simple exercises—such as flexing the leg on the thigh, the thigh on the abdomen, and so pressing the abdomen while taking a deep inspiration—was that it had done some genuine good, as, although the patient was by no means cured, she was very much better, and the heart was much less dilated than it was.

Nasal Disease Causing Neuralgia.

MR. COLLIER, in presenting a case of chronic neuralgia of a very severe character, tic of the face lasting four or five years, said the patient's teeth had been removed, the antrum had been examined, and everything had been done to find out the cause of the disease. The neuralgia was on the right side, and very severe. He examined the nose, and found on the right side a mere slot, which prevented the examination of the turbinated bones. By passing a probe he felt an obstruction, and examining further he came upon what was distinctly dead bone. Thinking the disease was due to that, he removed the growths and brought away a polypus as large as a Barcelona nut, and then brought away a large piece of bone, dead and covered with granulations

The patient was then put to bed and an alkaline lotion applied, and from that time had made excellent progress. The neuralgia had ceased, except for one or two slight relapses, due to the irritation caused by the healing process. The operation had taken place about two months and a half ago.

DR. CAGNEY, with the view of emphasizing the importance of being able to recognise a case of neuralgia situated in the nose, mentioned the case of a woman who had been to him for advice as to an operation. She said hat she had suffered such intolerable agony for twelve years that her life was a burden to her. He elicited from her that she had been to several hospitals and private doctors, and that she had taken drugs of every description. He tried some drugs which he thought might not have been used, but before advising an operation, which he thought would amount to an excision of Meckel's ganglion, he took the woman to MR. HORSLEY. MR. HORSLEY examined her, and said that such cases certainly warranted an operation, but that in his experience such an operation was not always satisfactory, and he recommended a constant current. He (DR. CAGNEY) found that a constant current applied every twenty-four hours absolutely subdued the pain. She had no pain whatever while it was being used ; but it should be remembered that such treatment could only be palliative. Some years ago the patient had been under the care of DR. HUGHES BENNETT, who had applied cocaine to the mucous membrane of the nose. On hearing this, DR. CAGNEY asked MR. COLLIER to examine her nose, and MR. COLLIER had found the conditions he had described in the case he had mentioned. The result was that the woman now lived a happy and comfortable life.

MR. COLLIER stated that in the case he had referred to there was a slight discharge from the nose, hardly noticeable. With regard to the case mentioned by DR. CAGNEY, it was undoubtedly a case that originated in the middle turbinated bone. The irritation of the nasal ganglion was transmitted to the Meckel's ganglion, and that ganglion was in such a state of irritation that the slightest stimulus was sufficient to set up reflex pain.

Resection of the Knee-joint.

MR. COLLIER also showed a case of resection of the knee in a boy. The case was so severe that the whole of the knee-joint was disorganized. The tibia could be moved from side to side upon the femur. There were two large sinuses, one on the inside and one on the outside. At first it was thought the limb would have to be amputated, but at the request of the boy's mother resection was performed. They were careful to dissect out the whole of the synovial membrane, to scrape away every part of the bone that seemed diseased, and to thoroughly drain the joint by passing in a very large drainage tube, and when the patella had been joined by a double silver wire, the leg was placed in a splint. The result was that the boy stood before them practically cured.

MR. BRODIE said that when he first saw the boy he thought it almost impossible to cure him by resection, and it must be very gratifying to MR. COLLIER to see such a successful termination to the operation.

Congenital Syphilitic Ulceration of the Tongue ; Nævus treated by Electrolysis.

MR. JACKSON CLARKE showed two cases occurring in children in the same family. The elder of the two children exhibited the rare condition of distinct and typical syphilitic ulceration of the tongue combined with interstitial keratitis. She had only one permanent tooth through, and it was of the typical Hutchinsonian appearance. There was a history of rash in the mother a few months before the child was born, and he had no doubt that it was a case of congenital syphilis. The patient had done very well on iodides. The younger child showed the result of treatment by electrolysis of a nævus of the nose. Electrolysis was very valuable as an aid either to remove a small nævus entirely or to reduce it to a scar before excising it. He reduced the nævus to a scar in order to prevent hæmorrhage when excising it. If excision became ultimately necessary it was made much less severe by previous electrolysis. The ordinary port wine marks might be treated by electrolysis also.

DR. CAGNEY said the present was one of those nævi in which the vascular tissue was very small and the stroma very considerable. He agreed with MR. CLARKE that the preliminary electrolysis might be done with the subsequent view to excision, especially in such cases as this. It had b en his lot to electrolyse some hundred or more nævi during the year, and he would be extremely dissatisfied if even a considerable minority were brought back for subsequent treatment by excision.

MR. COLLIER said he had seen a case lately where the electrolysis of a nævus on the upper eyelid had resulted in a distinct inability to raise the upper eyelid. In such a case the nævus should be at once excised. He would like to know how DR. CAGNEY would treat such a case.

DR. CAGNEY said everything depended upon the character of the nævus, nævi differing very materially in their constitution. He used electrolysis wherever it was practicable. He thought he had never seen a nævus implicating the upper eyelid alone. He had seen many about the orbit which implicated the upper eyelid, in which electrolysis had succeeded very well.

Progressive Muscular Atrophy.

DR. GUTHRIE showed a case of progressive muscular atrophy, which he believed also exhibited signs of incipient tabes. The patient was an accountant, 44 years of age. Three years ago he had broken his right arm while cycling, and had never quite regained power in the shoulder. Shortly after the accident he noticed weakness and wasting of the muscles in the right hand and forearm, and wrist-drop began to occur. Six months after the accident the left hand and forearm became similarly affected. Twenty-two years ago he had syphilis, which was followed by a rash and loss of hair. The muscular atrophy affected both forearms, and there was wasting of the long extensors and the extensors of the wrist. He had no power to oppose his thumb to his fingers with the left hand, though he could do it with the right hand. There was some affection of the serratus magnus, and also some affection of the supra-spinatus. With regard to the tabetic signs, the patient had the Argyll-Robertson phenomenon. When seen three months ago the pupils were unequal and contracted, and reacted slightly to light ; but very soon after, he (DR. GUTHRIE) found he could get no reaction to light. The patient was perfectly steady with his eyes shut, and not at all ataxic in his movements. For years he had suffered from lightning pains. He had also severe ab-

dominal pain, diarrhœa, and sense of constriction round his waist, which he attributed to a small dose of sulphur, but the symptoms were too severe to be caused in that manner. There was almost an entire loss of knee-jerk. His sensations were perfect everywhere. There was no reaction whatever of the extensors to faradism or galvanism. As regards the nature of the lesion he (DR. GUTHRIE) thought there could be little doubt that it was a case of syphilitic affection—not necessarily gummatous—of the anterior column of the cord in the upper dorsal and lower cervical regions, with probably a similar affection in the postero-external columns; and, therefore, a condition of muscular atrophy combined with tabes.

DR. GUTHRIE also exhibited a second case by way of contrast to the first. The patient had progressive muscular atrophy affecting only the muscles of the hands. The condition was not so severe as in the first case. He (DR. GUTHRIE) thought the patient had lateral sclerosis, because the knee-jerk was exaggerated, as were some of the other reflexes; otherwise he had no condition which really pointed to lateral sclerosis. The patient had advanced myopia, and also detached retina.

Erythema Nodosum.

DR. CAGNEY brought forward a case of erythema nodosum in a young woman. She suffered from flatulent dyspepsia. There was no rheumatic history, and no circulatory disturbance. For the last three or four months she had been troubled with a swelling in front of the upper part of the tibia on both sides, which was accompanied by heat and some tenderness. It was a wid ly diffused swelling, causing a certain amount of deformity in the appearance of the limb. It had the situation and character of an erythema nodosum, but differed from the ordinary description of erythema nodosum in its considerable size. It got better or worse according to the amount of rest the patient took. MR. COLLIER had seen the case, and agreed with him (DR. CAGNEY) that it did not affect the bone.

MR. COLLIER said he had examined the case, and he did not think the disease had attacked the periosteum.

DR. ABRAHAM had no doubt that the case was one of erythema nodosum, though it exhibited some peculiarities, and notably was on a larger scale than is usual. He recommended DR. CAGNEY to persist in the use of quinine and iron.

THERAPEUTICAL NOTES AND FORMULÆ.

Removal of Bones from the Throat.—Dr.
O. Schliep recommends drinking of some dilute acid to soften the fragment in cases where cocainization with the aid of the laryngoscope is impossible. Experiments with vinegar in Germany demonstrated that small fish bones are somewhat softened in 15–20 minutes. Small scales from shell-fish require one to two hours. He has also tried 1–5 per cent. solutions of hydrochloric acid

with and without pepsin. It is used as follows :— In case that the bone is situated high up, a stick, around the end of which is rolled a tuft of cotton, is dipped into a 2 per cent. solution (14 drops to 50 gms. of water) of hydrochloric acid and repeatedly pressed into the folds of the pharyngeal mucous membrane. In case that it is lower, or if the bone has passed into the stomach, this same solution may be frequently drunk.

(*Wiener Medizin Press.*)

Treatment of Eczema of the Eyelids.—
Dr. Trousseau has obtained unexpected results in this obstinate affection with local applications of a solution, without alcohol, of corrosive sublimate (1 to 4 gr. to 16 oz. of water). Compresses soaked in this fluid are also employed. Internally, one may give quinine, and locally employ a solution of carbolic acid to allay the itching. Salves are to be used only in the terminal period when desquamation of epithelium sets in, and then with caution; first trying those least irritating, as pure vaseline or lard, then bismuth, oxide of zinc, ichthyol, yellow oxide of mercury, or oil of cade.

(*Rev. Internat. Bibliog. Med.*)

A Simple Remedy for Cough.—Dr. Charles
E. Page, of Boston, says that the best remedy for cough he has ever found is a teaspoonful of moderately hot water, taken every time a paroxysm of cough comes on.—(*Medical Record.*)

Chronic Eczema of the Face.—Hebra re-
commends the following :—

R Salicylic acid ...	...	5 parts
Ichthyol	... ...	10 parts
Glycerine	... ...	10 parts
Alcohol (flavoured with		
peppermint and laven-		
der essence)...	...	40 parts
Rectified spirit...	...	60 parts

Sig.: To use as a paint.

(*Giornale di farmacia, etc.*)

Lemonade for Diabetics.

R Pure water	... ...	Oij
Pure glycerin ...	...	℥ꝰ–j
Citric acid	... ...	ʒi½

To be taken in small quantities within twenty four hours.

(*Journal des Praticiens.*)

A CLINICAL LECTURE

ON A CASE OF

POST-INFLUENZAL ARTHRITIS.

Delivered at the London Hospital, Dec. 13, 1894,

By A. ERNEST SANSOM, M.D., F.R.C.P.,

Physician to the London Hospital, and Lecturer on Clinical Medicine at the London Hospital Medical College.

GENTLEMEN,—I call your attention to an example of what I have called post-influenzal arthritis, but the articular disease is not the only circumstance of interest in the case. It is that of a woman, aged 46. She was admitted into this hospital five months ago, and she came complaining of pain chiefly in the arms and the knees. The family history is that her father died in a fit; but of her mother's history we know very little. She had one sister who died of phthisis, and one of her children was also consumptive, so that all we can say as regards hereditary predisposition is that, perhaps, there was a proclivity to nervous disease (on the side of her father) and to phthisis, because her sister and one of her children died of it. There was no traceable tendency whatever to rheumatism. When, however, we come to her personal history, we find she had rheumatic fever at 16 and 17 years of age, and a second time at 26, and she had had repeated subacute attacks since. There was no doubt, then, that she had rheumatic tendencies, and had indeed suffered from rheumatic fever on several occasions. The last illness of hers dated from her last confinement. She had ten children, and her last confinement was four months before she came into this hospital. That event was soon followed by a febrile ailment which might very easily have been mistaken for rheumatism. If you had asked her what it was she would have said she had another attack of rheumatic fever, but she happened to be attended by a doctor whom I know to be a careful observer, and who told me at once that it was not rheumatic fever at all, but influenza —he had no doubt whatever about it. At that time—at the time of what we will call influenza— she suffered pains in the knees, hands, and feet, and then, or very soon after, she had numbness and sensations of pins and needles in her fingers and toes. She said her feet felt cold and dead, and she had cramp in the calves of her legs. There were then arthritic pain, muscular pain, and disturbed sensations; but there was not the typical rheumatic swelling of the joints. When admitted she was well nourished, but we noticed that the muscles of her arms and legs showed signs of ill-nutrition. The arms were flabby and the interossei showed gaps, evidently they were somewhat wasted. She was a woman of ordinary intelligence; she had occasional giddiness and headache, and she complained of numbness in the fingers and toes, and of disturbance of sensations.

When we came to look at her more closely we found first of all, as regards her *motor power*, that when she was in bed she executed movements both of arms and legs, and of toes and fingers: but she was unable to walk. Her legs felt weak, she said, and gave way under her. Besides, we noticed she had disorderly tremulous movements in the fingers and in the hands, so that when she made any attempt to do anything with her arms and hands, there was a sort of convulsive movement, but not a very rapid one. Now we tested her in this way. First of all, we told her to bring the tips of her fore-fingers together in front of her with her eyes open, and in doing that she moved the muscles desultorily. Then we told her to shut her eyes and touch the tip of her nose, but instead of knowing perfectly well by her senses where her nose was, she went widely from the mark. You might say then that her movements were inco-ordinate, but it would not do to say that it was the inco-ordination of loco-motor ataxy. But let us go a little farther. We found that the muscles of the legs were rigid, and that those of the thighs and legs were set in action by mechanical stimuli. If we attempted to elicit the knee-jerks, for example, the muscles were thrown into a sort of clonic spasm, but if the question were put, " Did you elicit the knee-jerks or not?" we would say the knee-jerk was obtained with difficulty or not at all. Remember that this might be a false inference because the reason it was not elicited was that the dominant spasm of the muscle prevented it. If it were not for the spasm of the muscle it might be obvious. It is quite

enough to say there were no signs of ordinary locomotor ataxy. And we found there was no Argyll-Robertson phenomenon. There was a sufficient response by the pupil both to light and to accommodation. There was not that absolute non-response to light, although reaction to accommodation, which is called the Argyll-Robertson phenomenon, so that true locomotor ataxy was not present. And you see that locomotor ataxy could not explain all the symptoms, because there was an acute disease going on in an acute way—not a chronic one. Moreover, you could not explain by locomotor ataxy the spasmodic stiffening of the muscles. Then as regards the difference between the two sides, we found the left arm was rather weaker than the right, so that in the test with the dynamometer, the squeeze on the right side was 20 kilogrammes, while on the left side it was only 15 kilos., but of course the left is usually rather weaker than the right.

As regards *sensation* we found there was much tenderness on pressure of the muscles of the arms and legs; it was more in the left arm than the right, but to an equal degree in both legs. If we pinched the calves there was great pain; if we grasped the arms there was also much pain, while besides there were disordered sensations. There was numbness in all the extremities, and there were also impaired sensations to heat and cold. She called very hot water " a little warm," when applied to the left leg. Here we have then symptoms which, with pain locally, and with loss of power such as we have here, we might put down to neuritis; as you may find neuritis after alcohol, so you may get it after a number of infectious fevers, as well as after certain forms of poisoning. But neuritis would not explain all the signs we have here. You would not get the irritative condition of the muscles; quite the opposite, they would be flaccid, so that would not do to explain all. Then as regards the *nutrition*. There was wasting in the muscles of the limbs, especially the legs—both flexors and extensors.

As to *circulation*, when we examined the heart we found no notable enlargement of it, but there was a systolic murmur evident at the apex. This was inaudible on deep inspiration. We found also a systolic murmur at the third left interspace. This also was inaudible on full inspiration. The second sound seemed normally clear; it was not accentuated over the pulmonic semilunar valves. The inference was (considering that she presented

a long rheumatic history) that she had had mitral endocarditis which affected the mitral valve. The lesion was not considerable; if it had been so we should have expected a greater enlargement of the heart, more evidence of a strong tension in the pulmonary circuit (more blood thrown back on the pulmonary artery, and a loud pulmonic second sound), which was not the case. There was very little sign, except a murmur, which may be of little moment, at the apex of the heart. But the action of the heart was disturbed. She had frequent attacks of palpitation and pain in the region of the heart. Her usual pulse was 84 a minute; the tension was moderate, and the arteries were strictly normal, so far as we could make out, but attacks of palpitation occurred with attacks of pain.

As regards *respiration*, there was shortness of breath whenever she had these attacks—there was a paroxysmal dyspnoea—and then there was a cough and sometimes some blood streaked the expectoration. Yet we had no notable signs of anything wrong in the pulmonary tissue. What there was, so far as we could make out as regards respiration and the heart's action, was spasmodic and irregular.

Now we will consider the progress under treatment. We gave her the mixture of soda and gentian, and with it iodide of potassium, and we had the muscles of the limbs faradized every other day, and besides, we tried counter-irritation of the spine. There was an increase in the muscular power in the arm, and in three weeks the grip, as shown by the dynamometer, was 25 on the right side and 20 on the left, and in six weeks it had increased to 45 on the right and 40 on the left. It was evident that the muscles had gained a great deal of power. It was evident, too, that the movements of the right arm became better, but in the left the triceps jerk was exaggerated, and continued so for a long time. After, we tried to elicit the triceps jerk above the elbow by tapping, the arm being pronated—the muscles of the forearm and upper arm were set in action. So there was what is called " myotatic irritability," but her legs improved, and there was a great deal of motor improvement generally. She began to be able to walk—at first with help, and then afterwards without help. But there was always some rigidity about the muscles of the legs, and some spasmodic contractions of these; the muscles still remained tender and there was inco-ordination still manifest in some degree about the movements of the arm.

There was no abnormal rise of temperature all the time she was in the hospital. It was a non-febrile ailment. The urine was quite normal.

She was able to go out seven weeks after she came in, considerably improved.

Now let us think over the case. *First*, there was no doubt about one or two points. The patient had a rheumatic tendency and there was, though slight, some rheumatic valvular change in her heart. Now, the first signs of her illness were like those of rheumatic fever. There was again arthritis. We have had many other cases where influenza has been initiated by signs very like those of rheumatic fever—I do not know how you positively differentiate them at first. Pains in the joints, and fever, are signs that make you wait for a day or two before you are sure whether there is rheumatic fever or not. Remember that rheumatic fever is not the only acute disease occurring with pains in the joints. Cerebro-spinal meningitis is attended with like pains. But as the case goes on there are a good many symptoms that tell you it is not uncomplicated rheumatic fever. Influenza may complicate rheumatic fever. Some of the worst cases I have seen have been those of rheumatic fever complicated by influenza. I have seen a patient at the point of death—he did die soon after—with a clear history of having had the two diseases: acute rheumatism and then influenza. I have seen in young subjects, repeated attacks of influenza with most severe rheumatic fever and heart inflammation. So there is no doubt that influenza can complicate rheumatic fever, and it will also produce symptoms very like it. I have seen a case, without the slightest rheumatic proclivity, closely resemble rheumatic fever, and then a drop of temperature has taken place, with a complete loss of all pains and signs of rheumatism.

In this case, there was no lighting up of the old fires of rheumatism in the least degree. To differentiate from acute rheumatism, look at all those strange disturbances of sensation in the limbs, long-continued and non-febrile, the peculiar condition of the muscles, and the disturbed co-ordination of movements. The next question was, could we explain the signs by neuritis? As I said just now, undoubtedly influenza can be complicated by neuritis. Many observers have described neuritis as following more or less closely on influenza, but neuritis would not explain all. Spinal meningitis might, in considerable degree explain the symptoms both in the early history and later on.

Let us think a little about the pathogenesis of influenza. What is the disease due to? What are the causes of the symptoms? In the first place, there is no doubt that influenza is an infectious disease, and must obey the laws of other infectious diseases, and it must be due to like causes with other infectious diseases. It must be a microbe disease. We know that there are specific microbes that produce all the known forms of infectious disease. It may not be proved up to the hilt—it is not—but there is a very strong probability that the microbe that produces influenza is the one described by Pfeiffer of Berlin—one very difficult to cultivate, and difficult to get experimental evidence about. It is cultivated with great difficulty upon a soil that must contain hæmoglobin. The micro-organism is found in the air passages—in the pharynx, about the mouth, and all through the finer tubes. You must remember it is not the mere microbe that does the mischief in greatest measure. Proliferation may do something, but it is the poison the microbe produces that does the chief mischief. It is the toxin—the poison that is formed through the influence of the microbe that is the most important agency, if we are to follow the ordinary rules of evidence. The effects of that poison of influenza must be produced in the organism for a very long time—months and even years—after the attack. Many months after the initial attack of influenza you may get symptoms that are really to be referred to the original cause. Its early effect is fever—there is no doubt about that. We will not debate what the fever is, but at any rate it is a fever, resembling that produced by cerebro-spinal causes. Next, supposing we leave out, as debateable questions, how the microbes of influenza are diffused and propagated, we have an array of evidence that shows that the symptoms of influenza, long drawn out (both in its early and later periods) can be traced to disturbance of the vagus nerve. You may have signs of disturbance in all the areas subserved by the vagus nerve. You may get these in the respiratory tract—as spasmodic dyspnœa, or various pulmonary congestions. You may get it in the cardiac branches, for there may be an exceedingly quickened heart; or paroxysmal palpitations, or a very slow heart, or, between the two, an irregular heart. We find also symptoms referrible to disturbances of the branches distributed to the organs of the abdomen. You may observe many forms of dyspepsia, and in a large number the attacks are paroxysmal and intense.

Very often you observe all three areas affected together. There are attacks characterised by palpitation of the heart, by difficulty in breathing, and by paroxysmal dyspepsia, and that is what we may call a "vagus storm"—a storm in the area of the pneumogastric nerve. It would not do for us to say that the poison of influenza affected only the roots of the vagus nerve and passed to the branches, but we know very well that the evidence also shows that it fixes itself upon the upper part of the spinal cord, and often involves a great deal more than the upper part. We have observed very interesting cases of disease of the spinal cord after influenza. In one case I have seen it closely like cerebro-spinal meningitis, with retrojection of the head, stiffness of the muscles, extreme neck pains, and so on; and, as I told you, in the disease—cerebro-spinal meningitis—it is very common for the joints to be affected—they may be hot, red, painful, and swollen. In this case there was an affection of the joints that subsided and went away; it was an affection of the joints such as we might have in spinal cord disease. I do not think we need particularise the rest, but we will consider how the symptoms we have observed in this case would agree with those of a disease involving the spinal cord. We have symptoms consistent with disease of the antero-lateral columns:—muscular spasms and contractions, cramps (reflexes are hindered by the pre-existing spasms, but clonic spasms of the muscles are easily provoked). Then the grey matter of the anterior horns is probably involved, because we have motor paralysis and wasting; the posterior cornua, because we have delay in transmission of sensations and of thermic influences and painful impressions with various interferences with sensibility. Perhaps interference with the posterior columns, because there are inco-ordinate movements, however explained, as well as interference with the due transmission of sensibility of touch, pressure, and temperature.

The case was one of influenza in a rheumatic subject, but the influenza did not aggravate the rheumatism in any appreciable way. It produced symptoms that could be attributed to disease of the spinal cord, and perhaps of the peripheral nerves as well.

The patient was admitted again on September 28th, that is, about two months after her discharge, and six months from the commencement of the acute disease (influenza). She was admitted because her left knee had begun to swell, and became tender and painful. The signs then did not con-

form to those of rheumatism. Why? Because it was only *one* joint that was affected; rheumatism generally affects a number, it generally causes a definite fever, a definite swelling of the joints, subsiding in a limited time—not the lasting pains and intense congestion of osteo-arthritis.

In this case there was one joint specially affected—the left knee. The calf of the left leg measured 14½ in. as against 12¾ in. the right one. It was kept bent at an angle of 120, extension was resisted because of the pain, the tibia on the left side seemed to be slightly dislocated backwards. There was some evidence of fluctuation in the semiflexed position, and the patella was immovable. There was pain along the inner surface of the tibiæ and over the radius and ulna of the left arm; there was tenderness over both ankles, but no pain in these joints. There was no change in the heart conditions and no fever; the pulse was 66; so that rheumatism was excluded. As I have said, this differs from a rheumatic joint affection for the reason I have given, and if we look at the pathology of it, it is different. We ought to be very definite as regards rheumatism. Anything painful is called rheumatism, but it seems a very startling thing to say that rheumatism is only a slightly painful disorder. The pain of rheumatism is very limited. It lasts only a week or fortnight, and you do not expect a patient with rheumatism to have a return of the pain unless he gets a sub-acute attack. Then the effusion in rheumatism is limited. In many forms of non-rheumatic arthritis the inflammation of the joints is very much more intense than in rheumatism. You get enlargement of the ends of the bones, extreme congestion of the component tissues, the cartilages intensely inflamed, and the whole of the joint swollen and enlarged. Besides you get degenerative changes. There is a mixture, as it were, of intense inflammation and a rapid degeneration of the structures of the joints. So you get the bone infiltrated, the cartilage eroded, and the ends of the bone permanently altered; but you do not get anything like that in true rheumatism. There are many infectious diseases which are followed by non-rheumatic arthritis. Like changes occur in osteo-arthritis, what is commonly called rheumatic gout, though probably it has nothing to do with rheumatism nor with gout. But these changes do occur in various ways in disease of the spinal cord, and I take it that in all probability in this case the arthritic disease, like the arthritic pains at the

commencement, although those were only transient, was really due to some disease of the spinal cord. How produced it is difficult to say. A reasonable conjecture is that the centre that presides over the nutrition of the joints is in the area of the disease, and it becomes disturbed. In this interesting case, as in a large number of others which we have observed, where influenza has initiated the symptoms, the disease of the spinal cord is in not one of those definite forms you read of in text-books, but a disease, as it were, going over large areas.

Happily there is a better prognosis than in the special cord-diseases attended with sclerosis, for, under the influence of time and treatment, much improvement or even complete cure can take place. The treatment in this case was to keep the patient tranquil and well-nourished, to apply counter-irritation, and to keep the joint at rest by splint and bandage; then, under the influence of that complete tranquillity, which is the most important point of all, the knee resumes, as in this case, almost its normal characters.

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A CLINICAL LECTURE

ON TWO CASES OF

SARCOMA OF THE THIGH.

Delivered at University College Hospital, Oct. 29, 1894,

By CHRISTOPHER HEATH, F.R.C.S.,

Holme Professor of Clinical Surgery.

GENTLEMEN,—You will remember that I have had during the last three months two cases of amputation at the hip-joint, which is a somewhat unusual occurrence. The first case unfortunately happened during the vacation, and therefore many of you were not present at the time of the operation; but you will remember that you all saw the poor girl (for she lived until the week before last), and that I made comments upon the case when going round.

She was a girl, 16 years of age, and was admitted here on the 6th August. The first thing the patient noticed wrong with her left leg was that it felt stiff and ached a great deal. The pain was of a gnawing character and was worse at night time. That was three months ago. There was no swelling until two months ago, when a hard lump, which increased rapidly, was noticed in the upper part of the thigh, and the doctor was called in, and he attended her during the last month prior to her admission here. During the three weeks before admission, the tumour appeared to grow faster than when it was first noticed, and also to grow more in a downward direction. The leg also swelled during the last week or two. The patient had great pain in the thigh at times, and was obliged to have morphia and other drugs. She was a rather slightly built girl, and had wasted a good deal lately, having become very thin. The left limb was obviously swollen in its whole extent, and considerably more so in the upper part of the thigh. On the right side, $4\frac{1}{2}$ inches directly below the anterior superior spine, the circumference was 15 inches, and on the left side (that is the affected side, of course), $22\frac{1}{4}$ inches. The enlargement was chiefly on the anterior and outer aspect of the limb, and was caused by a large tumour in the muscles. The tumour felt hard and resistant, and nothing like fluctuation was perceived. The upper limit of the tumour was about 1 inch below the anterior superior spine on the outer side, while internally it had a convex border reaching up to a little above Poupart's ligament. Downward it reached to a point $7\frac{1}{2}$ inches vertically below the anterior superior spine, on the outer surface of the thigh. The posterior limit on the outside was marked by the anterior margin of the great trochanter. On the inner side the tumour was obscured by muscle, except close below the pubes, where it came almost to the surface. The back of the limb was free. Enlarged veins were to be seen crossing over the tumour, and a few large inguinal lymphatic glands could be felt on the surface, just over Poupart's ligament.

These are the principal features of the case. From the first it was quite clear that the only chance for the poor girl was to remove her thigh at the hip-joint. Accordingly, on the 11th August I amputated, taking every precaution to avoid hæmorrhage. I found I had to deal with an exceedingly soft tumour of the thigh, involving the femur and the muscles. The most marked feature in the case was that the growth in the muscles extended above Poupart's ligament and reached quite under the abdominal muscles, so that I had to remove the growth from beneath the abdominal muscles with my fingers in order to get it all away. As far as I could see, I did get it all away, and the patient rallied very fairly from

the operation; but I was quite sure, from the nature of the growth (it proved to be a well-marked round-celled sarcoma), that there would be a very rapid recurrence, and that proved to be the case. You will remember that by the beginning of October, when most of you had returned, there were already evidences of considerable reproduction of disease in the flaps. That went on increasing day by day until she died on the 20th October, when there were large masses of bluish-looking growth protruding from the cicatrix. In fact, it was a case which was practically hopeless from the first. Still, I thought it right to submit the patient to an operation, because there was just the chance that there might not be recurrence; at all events, it was better that she should die in that way quietly from recurrence and general giving way of the system, than that she should suffer from constant pain, owing to the presence of the tumour.

The case is very like one which I had in this hospital some years ago, and of which I show you a photograph (fig. 1). You will notice that it is very much the same sort of tumour, but the disease is rather lower down in the thigh. It was the case of a girl 16 years of age, who was admitted under my care on September 26th, 1887. She had enjoyed good health until six months previously, when a swelling was noticed in the middle of her left thigh. This swelling rapidly extended upwards and downwards. The patient was seen by myself nine weeks before admission, and amputation was advised, but the patient's friends preferred to try the powers of a herbalist, who treated the disease for about six weeks. During that time the growth increased very rapidly in size, and became very painful. For three weeks the patient had been confined to bed. On admission the whole of the left thigh was enormously enlarged, and, as you will see by the photograph, the superficial veins stood out as blue cords, some of which measured one-third of an inch across. The thigh was very tense, but elastic, and in some places distinct fluctuation could be obtained. At the upper limit of the growth, close to the groin, the circumference of the thigh measured 23 in.; at the middle and lower end of the growth it measured 25 in. The corresponding measurements of the sound thigh were $10\frac{1}{2}$, $11\frac{1}{2}$, and 14 inches respectively. The patient evidently suffered most severely from pain, which was, however, almost completely controlled by an injection of four minims of morphia night

and morning. The weight of the limb was so great that the patient was unable to move it. Her temperature ranged between 100° and 103.2°. There was some healthy skin in front, just below

FIG. 1.

the groin, and the buttock was not implicated by the disease, so I determined to remove the growth by disarticulating at the hip-joint.

The two cases were very much alike, except that in the more recent case of the girl Alice, the disease encroached more upon the groin than it did in the other case. In both cases I performed disarticulation at the hip-joint, and they both did very well for some time. The first girl went out of the hospital, and died at home some few weeks afterwards with recurrence in the muscles. Alice lived a long way from London, and I therefore preferred that she should stop on here. I told her friends, however, that she must eventually die, which she did in the hospital.

These are both examples of the round-celled sarcoma affecting the muscles. I consider this to be one of the most serious forms of sarcoma with which we have to deal. The only chance for the

patient is that the sarcoma should be dealt with in its early stage. If you can recognize the disease soon after it first appears, and then get rid of the whole of it, I think the patient has a chance of recovery. But when such cases have been allowed to go on, as both these were, for a good number of weeks before being submitted to treatment, it is practically impossible to get rid of the whole of the disease, and recurrence must take place, eventually ending in death. I do not, however, in either case regret that I operated, because I am quite sure that I gave very considerable relief for the time being.

Some years ago, I had another case of sarcoma of the muscles, which may be contrasted very well with these two cases. I have here a photograph of the patient (fig. 2), who was a boy, 16 years of

FIG. 2.

age, when he first came under my notice in 1883. It will be seen from the photograph that there was a very large mass of disease in the upper part of his right humerus. That disease, which had been noticed for about two years, was said to have followed a fall. The tumour involved the upper part of the humerus and scapula, and on the 4th July, 1883, I removed the whole of the upper part of the limb with the scapula and the outer third of the clavicle, as will be seen in the second photograph (fig. 3).

This last case contrasts very strongly with the other two cases, because it was of a very much more chronic nature. The distinguishing feature between the three cases was that in the boy there was ossification going on in the sarcomatous

growth; it was one of those cases of ossifying sarcoma which are very much less serious and deadly than cases of soft growth, which extend so rapidly through the muscles. I saw this boy at another hospital some months before he came under my care. He then refused to have anything done, and went away. Then he happened to come under the notice of an old pupil of this hospital who eventually persuaded him to come back to me in June, 1883, some six months afterwards, with a view of having an operation performed. I have in this bottle part (the other part is in the museum of the College of Surgeons) of the growth; and you will see what an enormous mass there is connected with the humerus, also involving the scapula as well, and I have here a drawing which illustrates the point exceedingly well. You will

FIG. 3.

see a section showing the line of the humerus and the great mass of disease attached to it, involving mainly the muscles, and you will see a great mass of disease connected principally with the venter of the scapula. The boy made a perfectly good recovery, and went home; but he came back again more than once. The growth was a spindle-celled sarcoma undergoing ossification, and, some months after, he came back and I removed a nodule from the cicatrix. Again, 2½ years after the original operation, when he had a recurrence in the pectoral muscle, I cut a large mass out of it. He died in January, 1891, exactly eight years from the time when I first saw him. You will therefore see what a very different history it is from that of the others, although these are all examples of

sarcoma. In this lad it was a comparatively slow pro-
cess by which he died, whereas in the two girls it
was about as rapid as any form of cancer could be.

Now let me take the other form of sarcoma
which is directly connected with the bone itself,
and only involves the muscles very slightly and
secondarily. We have a good example here in
the preparation from the patient whom you saw
just now doing so well in private ward No. 1—
the man whose hip I amputated last Wednesday.
I will give you shortly the notes of the case. The
patient was admitted on October 20th, 1894. He
is about 24 years of age, and was employed as a
telegraphist in the Government Telegraph offices.
About four years ago he noticed a slight pain in
the hip-joint when walking. It lasted two or three
days, and then completely disappeared. Six
months later it commenced again, and lasted
longer, disappearing after a week. This occurred
again, and each time the pain lasted longer. The
last attack occurred last Christmas, and lasted
about a month. It is a little difficult, I think, to
connect those pains with the tumour, but it is
conceivable that even as early as that there may
have been some growth about the periosteum,
which may have produced pain. At the end of
July he had an attack of pain, and in the first week
of August he went down to Aldershot. There
one night the pain came on much more severely
than usual, preventing him from sleeping. On
the 19th of September he was obliged to give up
his work. During the last month he had noticed
that there had been a considerable swelling round
the joint. He had seen doctors, who gave him
medicine and liniments to rub the joint with.
Five weeks ago he went to another hospital, where
they told him he was suffering from sciatica. The
thigh was not examined, however, but he was
ordered to blister it. When he was eventually
sent to me the length of the tumour vertically
was from six to eight inches, but the lower margin
was ill-defined, and therefore the exact limit was
doubtful. It was firmly fixed, and the muscles
could be felt stretched over it. It did not reach
to the level of Poupart's ligament, and no fluctua-
tion could be felt at any point.

When I first saw the patient at my own house,
I at once recognized the very grave nature of the
case, there being a tumour evidently connected with
the upper part of the thigh, which from the history
was a sarcoma, and was presumably a periosteal
sarcoma. Let me say that the sarcomata con-
nected with bone are divided into two great
classes. First, there is periosteal sarcoma, which
springs from the periosteum, and grows outwards,
not involving materially the bone itself, but growing
entirely on the outside of it. Secondly, there is
the form which is the endosteal, and generally of
the myeloid variety, which expands the bone, as
will be seen in this bottle, which contains a section
of the head of the tibia, expanded by a myeloid
growth, from a case which Mr. Beck amputated
a few years ago. These two classes differ most
materially as regards their nature, because the
periosteal sarcomata are invariably spindle-celled;
whereas the sarcomata which involve the interior
of bone are generally myeloid, and you probably
know that a myeloid or large-celled sarcoma is
much less deadly than the spindle-celled variety.
We find cases of myeloid sarcoma particularly
about the lower jaw, and if the sarcoma is cut
freely away, it often gives no further trouble; but
where there is a spindle-celled sarcoma there is, I
believe, only one thing to be done, namely, to
get rid of the entire bone. Even if you have a
periosteal tumour at the lower end of the femur
and venture to amputate through the shaft, you
will almost invariably have recurrence in the
stump; and certainly when you have a spindle-
celled sarcoma involving the upper part, as high
as we have in this preparation, there can be no
question in any surgeon's mind that the only thing
is to get rid of the entire bone.

Having then to deal with a periosteal sarcoma,
as I believed, and as it eventually proved to be, I
told the patient at once that I thought the only
chance of saving his life would be to remove his
limb, and he had the good sense to agree to what
I proposed. I told him I would take him into the
hospital, and do what was necessary for him. Still,
you know, there is a good deal of doubt about all
these cases. Whatever your experience may be, I
think you should not venture to embark on such
an important operation as disarticulation at the
hip-joint without making quite sure that you have
diagnosed the case correctly. Let me remind you
that when we went round the hospital one day I
inserted an aspirator-needle in order to make sure
that there was no fluid, and I may further remind
you that at the time of the operation, before I
began to disarticulate, I took a scalpel and cut
well into the tumour down to the bone, in order to
make quite sure that it was a solid growth and
connected with the bone. Ascertaining in a few

seconds that that was the case, it only remained for me to amputate at the hip-joint; but before speaking of the operation itself, let me just say with regard to this patient that I have good hopes that he may have no further trouble. Of course it is too early to be sure about that; but considering that I have removed the entire tumour and the bone from which it springs, I venture to hope that he will be saved further trouble; and although, of course, he will be mutilated by the loss of his limb at the hip-joint, he will be able to get about with a crutch, and eventually, with the aid of an artificial limb, will be able to resume his work at the Telegraph Office.

With regard to the operation, I said at the time that we took every precaution to avoid shock, because shock is the thing that we have to fear in cases of large operations, such as amputation at the hip-joint. If you look into the literature of the subject, you will find that the mortality after operation at the hip is exceedingly large. One great improvement was made some years ago by Lister, when he introduced a very large screw tourniquet, which is generally known as Lister's abdominal tourniquet. If that instrument is applied properly over the abdominal aorta, putting the pad just below the umbilicus, you can compress the abdominal aorta for some minutes without doing any harm, and in that way control the enormous rush of blood through the femoral artery. In addition to that we took the precaution, as you will remember, of putting an Esmarch's bandage over the doomed limb, and we left it on. There was no object in taking it off again; the limb had to be taken off, and it could be taken off just as conveniently with the bandage on as without it. Had it been possible I should also have employed Esmarch's elastic cord. It is quite possible, with a little contriving, so to apply Esmarch's cord that you may make compression of the femoral artery at the groin, provided, of course, you do not want to go quite up to that point. Where, however, you have to go quite up to the hip-joint, I do not think it is feasible to employ it. Lately, in the *Lancet*, I noticed a paper by an American surgeon named Wyeth, in which he proposes to get over the difficulty which we all have experienced in keeping the cord in its right position, by an ingenious insertion of long pins, which are pushed through the limb just at the point where the cord goes. Then you will notice that he proposes to carry the incision quite

down to the bone, and to dissect the tissues up, making another incision along the side after encircling the limb, very much in the manner which was originally proposed by a Birmingham surgeon —Mr. Furneaux Jordan—and which is an exceedingly good method of amputating the hip-joint in suitable cases. This, however, was not a suitable case for that kind of operation. If you have to amputate for tumour in the middle of the thigh, or even in the lower part of the thigh, and choose to amputate through the hip-joint, I know of no better method than that of Mr. Furneaux Jordan, because by means of a circular cut you are enabled at once to take up and tie the femoral vessels, and you can, at your leisure, dissect up all the tissues and disarticulate, and, having sawn the femur through low down, you disarticulate easily enough. That is not the case, however, where you have a tumour high up, as we had here. It would have been quite impossible to have done the operation that I wanted to do by the method described.

Without in the least cavilling at this gentleman's proposal, let me notice one or two points. The time occupied in his operation was fifty minutes. Now, I very much object to operate against time, and I should never think of asking any gentleman to take a stop-watch out of his pocket, and see how long I was about an operation. I am told, however, by my house-surgeon, that a gentleman did take the trouble to time my operation the other day, and he found that I cut the limb off in something like 4½ minutes. Now, between four or five minutes and fifty minutes there is a very great difference, and I am sure that my patient was in the theatre nothing like fifty minutes altogether. When you read of prolonged operations of this kind, which take fifty minutes to do, you can hardly be surprised to hear that two hours later the pulse went down, and that the patient sank and died twenty-eight hours after the operation. I do not want to criticise American surgery, but I must say I think it is more satisfactory to do an operation a little more rapidly, and keep your patient alive.

There are one or two other points in connection with my case that you may remember. I marked out my flap, and then I cut down upon the femoral artery and the vein and tied them separately. I look upon that as a very important thing to do. Notwithstanding that Mr. Johnson kindly took charge of the Lister tourniquet, I would not trust

to that alone, but cutting down upon the femoral artery with the amputating knife, I tied the femoral artery and vein, as you remember, and had no further trouble with them. I did that both in the case of the girl I operated upon in August and in other cases with great advantage, and I strongly recommend you to adopt that method, as the minute or two lost while you pause to tie the femoral artery does not make the least difference to the operation, and it gives you the certainty that you have your main vessels perfectly secure. Then I disarticulated, as you know, and I cut my flap from the healthy buttock, as well as from the front. You will have observed now that the flaps came exceedingly well together, and that the patient is making as good progress as possible. I may say that there was a little suspicion that I might have gone a little too close to the anterior edge of the tumour. I did not think that I had done so, but you may remember that with the scissors I cut away some of the superfluous tissues and muscles, which I thought might by some possibility have become affected by the tumour, and in that way make doubly secure.

Again, let me remind you that in this case, as in all other large operations, I adopted the plan of injecting hot brandy and water into the rectum before the patient was brought into the theatre. I think that is a very important thing to do. Two ounces of brandy with four ounces of hot water thrown into the rectum make a very useful stand-by for the patient, and certainly prevent his getting faint during the operation. Let me remind you also that after we had secured all the vessels and stitched the wound up, we still kept the opposite leg raised, so as to throw as much of the patient's blood as we could into his body, and particularly into the brain, and he was carried back to bed in that position. Fortunately, his ward is on the same floor as the theatre. But supposing that the patient had been a woman, and that we had been obliged to carry her upstairs, I should have directed the porters to reverse the mattress on which she was carried, so as to carry her head downwards. I mention this because I think it is of considerable importance, that in carrying patients upstairs after a severe operation they should be carried with their heads down. In that way you will find that you prevent their getting faint, and they arrive at the top of the staircase better than when they started, instead of being rather worse as they otherwise would be.

Let me say one word with regard to the other variety of tumour—the myeloid tumour, of which I do not happen to have had an example lately in the hospital. The specimen in the bottle illustrates it extremely well, as does also this drawing from Mr. Hutchinson's "Clinical Surgery." In the one case you will notice the tibia and in the other the femur, but the same features are common to both, namely, great expansion of the bone by the myeloid, soft growth in the interior, and also the development of cysts, which is a very common feature in these myeloid tumours.

It happens that at this moment I have under my notice, in private, a young lady who I believe has a myeloid tumour of the femur. I have seen her once or twice in consultation with her own doctor, but we have not yet ventured to say to her or to her friends what I am sure will have to be said to them eventually, namely, that she must submit to amputation of the thigh. She is a young woman of 30, and has a soft tumour at the lower end of the femur, which gives the characteristic crackling of bone when you press upon it. That, I think, is most distinctive of a myeloid growth. The growth begins in the interior of the bone, and thins all the bone around until the bone crackles like an egg-shell. When you feel that crackling you may be quite sure that there is some growth inside the bone, and the probability is, if it is of a fairly rapid character, that it is a myeloid growth, and the sooner the limb is amputated the better.

Let me also call your attention to the fact that the articular cartilage is not encroached upon, as you will notice in the drawing and also in the preparation before us. It is a common and constant feature of these myeloid growths that they push the articular cartilage before them into the interior of the joint, but the joint itself is never involved; whereas if you have to deal with a cancerous growth of the lower end of the femur, you will find that it very soon breaks through the cartilage and involves the interior of the cavity.

Now comes the question of amputation. Would it be necessary in a case of a myeloid growth of that kind at the lower end of the femur to amputate at the hip-joint? Certainly not. All you have to do is to be careful that you amputate at a point well above the growth, so that there can be no possibility of any portion of the growth being left to induce recurrence. If you amputate, say in the middle of the thigh, or even in the lower third, you will be quite safe, and it is a great thing, if

possible, to save the patient a part of the thigh, because, in the first place, the danger is very much less than in amputation of the hip-joint; and secondly, an artificial limb can be much more easily adapted. I hope that my private case will not lead to anything more serious than amputation of the thigh, and I will take an opportunity, if it should come to that, to bring the specimen here one day, so that you can see in a recent condition what a myeloid growth is like to the naked eye. I need hardly say that microscopically there is no difficulty in identifying it, because you find numerous large multi-nucleated cells which are so characteristic of the disease.

Postscript.—The man with sarcoma of the thigh made a good recovery from the operation, and left the hospital at the end of six weeks. Three weeks later he returned with evident recurrence of disease on the left innominate bone, and the right pleura full of serous fluid, which was aspirated.

TYPES OF IDIOCY AND IMBECILITY.

BY

FLETCHER BEACH, M.B., F.R.C.P. Lond.,

Physician to the West End Hospital for Diseases of the Nervous System, Paralysis, and Epilepsy; Honorary General Secretary Medico-Psychological Association; Formerly Medical Superintendent of the Darenth Asylum for Imbecile Children.

PARALYTIC IDIOCY AND IMBECILITY.

CASES of this kind may be congenital or acquired in character. The former condition, which will now be considered, is usually due to an inflammation of the brain or meninges during fœtal life, or to porencephalus. Sometimes only one side of the brain is affected, and there is atrophy of that side with coexistent atrophy of the limbs on the opposite side of the body. Though imbecility is not necessarily the result, yet examples of the disease are of fairly frequent occurrence. According to Van der Kolk, everything depends upon the more or less healthy state of one hemisphere of the brain. "If," he says, "as from the nature of the case seldom occurs, the inflammation

and affection of the pia mater has not extended to this hemisphere, and if the grey matter under the cerebral convolutions has here continued perfectly sound, there is no reason why this remaining hemisphere should not be able to act without impediment in the exercise of those functions which are necessary to our mental powers, just as one eye sees as sharply though the other be lost. But where grey matter is injured in both hemispheres, particularly anteriorly, disturbance of the intellectual faculties will be inevitable." Generally paralytic imbeciles make fair progress mentally under training, but physically—that is, with regard to the paralyzed limbs—little improvement ensues. However, massage and galvanism of the atrophied muscles may be tried in the hope of improving their nutrition, but it is necessary for the attainment of a good result that this treatment should be continued for some years.

With reference to porencephalus, there are two kinds—a true, and a false. The former is congenital in origin, and is characterized by the existence of a communication between the lateral ventricle and that part of the convex surface which is implicated. False porencephalus, on the other hand, comes on after birth, and is due to softening, hæmorrhage, or inflammation of the brain substance; usually there is no communication with the lateral ventricle. This latter kind of porencephalus does not come into action in the congenital variety of paralytic idiocy which we are now considering. True porencephalus has been said to be caused by arrest of development, hydrocephalus, and profound anæmia of the convolutions bordering the Sylvian fissure. In this kind the borders of the *porus* are not hollowed perpendicularly, and the neighbouring convolutions are reflected along their walls, going from the convex surface to the bottom of the *porus*. The following are illustrative cases:—

W. B., aged 9 years on admission, was a fairly nourished boy, whose parents were temperate and healthy people, not connected by consanguinity. When the child was born, his right side was found to be powerless, in consequence, as is supposed, of the mother during pregnancy falling down and striking her left side with violence against a wall. On admission, his head was found to be flattened on the left side, but of fair size, being 19¾ inches in circumference. He limped with his right foot in walking, and the fingers of his right hand were flexed in the palm. He could read a few letters,

print with his left hand "a" and "o," count to 20, and knew a few colours. He made fair progress in school, so that, after two or three years, he was able to read from the First Standard, write and transcribe with his left hand in a book, work easy addition, subtraction, and multiplication sums, and recognize all the colours and shades. He made much more progress as time went on, and was sent to the tailor's shop. There he progressed equally well, and as a result of remedial measures, he was able to straighten the fingers of his right hand and make use of them in dressing himself and in performing his domestic and industrial work. In cases similar to this, not only do the muscles atrophy, but there is shortening of the bones on the affected side. Thus, on the right arm (affected side) the measurements were :—

From point of acromion process to external condyle of humerus, $10\frac{1}{2}$ inches; from external condyle of humerus to styloid process of radius, $8\frac{1}{4}$ inches; from centre of wrist to end of middle finger, $6\frac{5}{8}$ inches; circumference round centre of deltoid, 8 inches; circumference round centre of biceps, $7\frac{1}{2}$ inches; circumference round forearm $1\frac{1}{2}$ inch in front of inner bend of elbow, $7\frac{1}{2}$ inches; circumference round forearm 1 inch in front of wrist, $5\frac{5}{8}$ inches.

On the left arm (unaffected side) the corresponding measurements were $11\frac{1}{4}$, $8\frac{3}{8}$, $7\frac{3}{8}$, $9\frac{1}{2}$, 9, $8\frac{3}{4}$, and $6\frac{1}{2}$ inches respectively. Every measurement of the affected arm is seen to be less than that of the unaffected one, and so it was with the leg.

G. L., aged 10 years on admission, was born with the left arm contracted and useless, and little

power in the leg, though he could move them about. The imbecility was supposed to be due to a fright of the mother during pregnancy. The father was intemperate, and at times suffered from severe headache. The paternal aunt was epileptic. The child could not speak, but cried when he wanted anything; he was subject to fits, and his mental capacity was very small. At his death, which took place six months after admission, there was found to be a space between the dura mater and the cerebrum, filled with fluid, taking the place of the brain which had undergone atrophy.

SPORADIC CRETINISM.

This type is fairly common in this country, and patients affected with it bear a marked resemblance to one another, as regards their bodily conformation, facial physiognomy, mental characteristics and the symptoms generally. They have usually a dolicocephalic head, which is large in proportion to the size of the body, flat at the top, spread out at the sides, often narrow in front and broad behind; anterior fontanelle open sometimes to an advanced period of life; sparse, coarse, dry hair, like horsehair, usually of a brown or black colour. The forehead is low, narrow, and depressed laterally. The eyelids of a pale, bluish hue, are swollen and infiltrated with a solid œdema which does not pit on pressure. The subcutaneous tissue of the hands and feet is similarly affected. Nose pug-shaped, cheeks full and flabby, mouth large, lips thick and often slightly apart, through which an enlarged tongue occasionally protrudes. Teeth frequently carious and implanted irregularly; the second dentition is either deficient or completed at an advanced age. Chin small, with often a layer of fat beneath it, forming a double chin. Ears large, but not malformed; neck short, thick, presenting usually no sign of a thyroid gland. Well-developed fatty swellings are seen in most cases on each side of the neck, and sometimes in the armpits and other regions; occasionally they disappear before death. The abdomen is large, distended, and contains a quantity of subcutaneous fat; frequently there are umbilical and occasionally inguinal herniæ. There is arrested development of the genital organs; the testicles are small, and descend late, and the uterus and ovaries are often entirely undeveloped. The menstrual function is either not established or is irregular; there is no sexual appetite. The arms and legs are short and curved,

but this condition is not due to rickets ; the hands and feet in some of my cases, though small, were broad, thick, blue, and usually dry and scaly. The skin of the body, as well as the upper and lower extremities, is thick, easily separable from the muscles, whitish in colour, dry and rough, owing to the almost entire absence of perspiration : on the face it is yellow or waxy, giving rise to a sallow appearance. Digestion is good, as these patients eat slowly ; but there is always constipation, some-times obstinate in character. Respiration is often constrained, and the breath has a disagreeable odour. The pulse is small and feeble, the tem-perature taken by the rectum subnormal, and hence there is great susceptibility to cold. The urinary secretion is normal, but there is usually a deficiency in the amount of the urea excreted. The voice is rough, hoarse, harsh or squeaky, the gait clumsy and waddling, and there is often great repugnance to making any movement. General sensation is normal, and the special senses, with the exception of smell and taste, prove to be well developed. The knee-jerks are usually present, and the plantar reflexes normal. Speech is gene-rally limited to a few words, often monosyllabic, and consisting of "Yes" and "No; usually they can say their names. As far as the intelligence goes, some are low grade idiots ; others at 25 or 30 years of age have the mental capacity of a child of 4 or 5. They have the power of observation, imitation, and attention, and remember faces ; but they cannot dress or wash, or, until trained, feed themselves without help. They are good-natured, placid in temperament, and fond of those who have to attend upon them. The duration of the disease is variable ; some die in infancy and early childhood, others live to an advanced age. Death is due to convulsions, bronchitis, pneumonia, diarrhœa, or simple exhaustion. The disease is due to absence of function of the thyroid gland ; of sixteen autopsies, of which I have been able to find an account, eight having been made by myself, there was no thyroid in fourteen, but a bronchocele in two.

This is not the place to allude to the theories of the absence or disturbance of function of the thyroid gland, but there is no doubt that this is the cause of sporadic cretinism. The recent treat-ment by thyrodin tablets has proved markedly efficacious in the improvement of these cases, although, as in myxœdema, the patients must undergo the thyroid treatment during the whole of

their lives. The following are cases of sporadic cretinism :—

F. R., aged 5 years on admission, was the child of healthy parents, who were not connected by consanguinity. The father was very intemperate, and was so even at times of coition. The maternal grandmother suffered from melancholia for six weeks at the change of life, and the mother was subject to neuralgia. She ascribes the child's condition to fright and trouble during pregnancy. The child was born in London, had always been well fed, and the situation of the house was said to be airy. This was the eldest child ; two others had been born, one of whom had died, but they presented no malformation. On admission, she was seen to be a well-marked case of sporadic cretinism ; was well nourished, of fair complexion, with brown hair, 2 feet 6½ inches in height, and weighed 20 lbs. Her head was of an average size, flattened at the top and spread out at the sides. It measured 20¼ inches in circumference, 11 inches transversely, and 11¼ inches antero-pos-teriorly. A depression could be felt in the region of the anterior fontanelle. Her face was broad. Her forehead measured 4 inches transversely, and there was a distance between it and the chin of 5¼ inches. Complexion sallow ; nose pug-shaped ; mouth usually open, and tongue protruding ; lips thick ; arch of palate flattened ; teeth regular. No goitre present, and apparently no thyroid gland, but fatty swellings in the posterior triangles of the neck. The arms and legs were short and curved. Her skin was much smoother than usual, and could be easily separated from the subjacent tissue. Her countenance was usually vacant, but she would often smile when spoken to, although she could not speak. She was of a placid and quiet disposition. She could not stand nor walk, but spent most of her time in a chair. At the autopsy, the brain weighed 38 ounces, and the convolutions were of normal size. The trachea showed no sign of a thyroid gland, but in the posterior triangles of the neck were two fatty tumours, small in size, pinkish in colour and lobulated, without any investing envelope, and sending processes upwards and backwards beneath the sterno-mastoid muscles and downwards beneath the clavicles.

M. A. H., aged 15 on admission, was a well-marked case, but, unfortunately, I was unable to obtain any family history. She was a well-nourished girl of muddy complexion, 37 inches in height. Her head measured 19 inches in cir-

cumference, 11½ inches transversely, and 12½ inches antero-posteriorly. The hair was dark in colour, coarse and thick. Her face was broad, and her aspect pleasing; she was cheerful and good-tempered usually, but could not bear to be laughed at, and was sulky at times. Eyes hazel, nose pug-shaped, lips thick and generally slightly apart, teeth in fairly good condition, arch of palate case any fatty tumours in the posterior triangles of the neck. She had good use of her limbs, but was very slow in her movements. Her special senses, especially sight and hearing, were well developed. She could talk, but not fluently, and her voice was thick and husky. Her powers of observation, imitation, attention and memory were very good. She was of an affectionate disposition,

normal. Her arms and legs were short and slightly curved, and her hands and feet very thick and broad. The skin of the body was thick, and over the arms and legs easily separable from the subjacent muscles. She was exceedingly stout, and not only had the so-called "double chin," but a considerable amount of fat in the abdominal walls. Her neck was short and thick, no thyroid gland could be felt, but neither were there in this very fond of her nurse and of another cretin, a boy about her own age, who was in the same ward. She considerably improved during her residence at Darenth, became more intelligent, and took more interest in what went on in the ward. She learnt to wash, dress, and feed herself. She menstruated irregularly. The photograph represents her at the age of 24.

(To be continued.)

CLINICAL CASES WITH COMMENTS.

Read before the Folkestone Medical Society, Nov. 28, 1894,

BY

W. JOSEPH TYSON, M.D., M.R.C.P. Lond.,

Senior Medical Officer, Victoria Hospital, Folkestone.

GENTLEMEN,—A short time ago while looking over my old note-books, it occurred to me that a paper detailing some of the more interesting cases with comments, might possibly be useful. And here may I add a word on the desirability of early note-taking, as the recollections of diagnosis, prognosis, and treatment—and of the mistakes— may be of practical utility later. The first case I will describe is one of

Acute Periostitis of the Tibia, death occurring in ten days from Pyæmia.

A. B., aged 7 years, living in Folkestone, on Thursday, January 18th, 1877, had a fall, and hurt his left tibia ; on the following Friday and Saturday complained of pain in the leg. I saw him for the first time on Sunday evening, January 21st, his temperature was 104° ; the left leg red, very hot and tender. He was ordered to bed.

Monday and Tuesday following the temperature remained high, and on the latter day some vesicles appeared, but no fluctuation in leg detected ; for the rest of the week he was exceedingly low, but took a fair amount of nourishment, such as eggs, beef-tea, and milk, and during the last three days of the week a little brandy. On Sunday afternoon, January 28th, the inflammation in the leg was apparently gone, in fact, the last three or four days the leg looked almost well, but unfortunately I had not examined it minutely during those days until that afternoon, when I made out a boggy feeling on the anterior surface of the bone ; in pushing in a needle some pus escaped ; I then laid bare the tibia for three inches ; the periosteum was separated from the bone to that extent, and between them was a large quantity of caseous, purulent material, some three or four ounces. The cavity was washed out with carbolic acid lotion, and carbolic acid oil applied.

The next morning the boy died at 9 a.m. On the preceding Thursday and Friday the patient had suffered with diarrhœa.

Post-mortem.—The kidneys showed multiple abscesses varying in size from that of a hemp seed to that of a hazel nut, occupying mostly the surface of the kidney.

Liver. Three or four abscesses, not larger than a mustard seed, were seen on the upper surface, but none in the interior of the organ.

Heart. At the apex was found a yellowish, and rather firm lump, the size of a small Spanish nut, and breaking down in its centre ; another, similar in character, was in the wall of the left ventricle.

Lungs. The base of left lung was consolidated, and there were multiple abscesses the size of peas running throughout it ; only two or three abscesses were seen in the right lung.

There was no pericarditis, no pleurisy, no peritonitis present. No pus was found in the soft parts of the affected leg. The articular ends of the tibia were unaffected.

The following remarks were appended to my notes at the time : " This was a typical case of periosteal abscess or acute necrosis, followed by pyæmia, corresponding almost exactly to the classical cases described in pathology.

" The temperature was high throughout. I took the case at first to be one of erysipelas on account of the skin being so rapidly inflamed, but as the temperature remained high whilst the inflammation of the skin subsided, the case was one in which the bone was more likely affected than the skin."

Remarks. The presence of an eruption in connection with the septic condition, variously called " acute periostitis," " infective osteo-myelitis," " malignant periostitis," " septic osteitis," " periosteal abscess," or " acute necrosis," etc., is not very common, and mistakes have been made in the early diagnosis of the above kind of case ; the eruption is generally of erysipelatous or erythematous character, and accompanies the early stage of the disease.

I may also mention here that many of these cases have been diagnosed as rheumatic in their commencement on account of the pains occurring in the joints in the neighbourhood of the septic mischief. It is necessary, then, when any extensive erysipelatous eruption appears over the surface of a bone to think of a deeper and more serious inflammation occurring in the bone itself.

Pyæmia, resulting from compound comminuted fracture of the tibia, the skin being only slightly broken.

Mark N., aged 37, was admitted into Folkestone Infirmary on May 6, 1878. The case went on

well for the first fourteen days; he then complained of pain at lower part of right chest, the pain lasting off and on for about twelve days. On Monday, June 3, when sitting up in bed, he fell back and died.

Post-mortem.—On under surface of base of left lung, a pyæmic patch one inch square attached to the surface of the diaphragm. Right pleural cavity contained three pints of fluid, and the pleura covering the lung was rough and covered with recent lymph; at the base of the lung was a patch similar to that found in the left lung. The heart and liver and left kidney healthy.

Comments. The immense importance of antiseptic or aseptic surgery was not fully realised at this date. The wound was a small one, which did not at the time attract much notice, and the ward was exceedingly small and overcrowded.

The time is coming, if it has not already come, when all compound fractures will be treated actively by uniting the two ends of the bone, and then thoroughly irrigating the wound as soon as possible after the accident; or, if this is not done, then encasing the limb in a Croft plaster splint. Both methods practically fix the ends of the bone, whereas, by the ordinary three splints this is impossible to do satisfactorily.

Pyæmic Ostitis of the Upper Jaw ; Sloughing of the Soft Parts, etc. Death.

Mrs. W., aged 67, living in Folkestone, was seen on March 25th, 1879, when the right third molar (wisdom-tooth) was extracted on account of the extreme pain extending from right side of the face to the head; the extraction gave no difficulty. Soon after a small slough came away, the cavity remaining being about the size of a small bean; this cavity went on enlarging until nearly the whole of the mucous membrane of the upper jaw sloughed away; the right side of the face became much swollen, the right eye pushed out. The patient died exhausted on April 30th, a month after I first saw her.

Many years ago I remember reading of one or two similar cases to the one I have just described, but they are rarely met with. Some acute inflammatory septic condition starts round a tooth, generally a molar, and this is of such a violent character that the surrounding parts rapidly slough, and the patient is quickly poisoned. These cases seem analogous to those diseases occurring in children, known as noma valvæ and cancrum oris,

and again to that form of carbuncle, called "facial carbuncle," which occurs for the most part in young men with such fatal results.

(To be concluded.)

THERAPEUTICAL NOTES AND FORMULÆ.

Treatment of Piles by Kosobudki's Modification of Unna's Treatment by Chrysarobin.—Dr. J. McLeod relates the case of a patient, aged 40, who had suffered from internal hæmorrhoids for twelve years. They used to prolapse every few weeks, and especially if he indulged unduly in alcohol. He says he got relief only when they "burst," causing loss of a considerable quantity of blood. Then they did not trouble him till the next prolapse.

In January, 1893, he began using suppositories made in accordance with the following formula :—

R.	Chrysarobin	... ...	gr.j
	Iodoform	... ...	gr.¼
	Ext. belladon.	... ...	gr.⅛
	Coca butter	... ...	gr.xxx
	Glycerine	... q. s.	ft. suppositor
	Cocain hydrochlor.	...	gr.¼

He used altogether 3½ dozen of these—one daily. The hæmorrhoids gradually diminished in size from January till May, at which date they disappeared, and he has had no return since then. He can indulge in drink freely without inducing an attack.

An important adjunct to the treatment was the "squatting" position at stool, which he was induced to adopt. He has gained in weight, and feels better in every way.

(Austral. Med. Gas.)

Tænifuge for Adults.—

R.	Ethereal ext. male fern		f ℥iss
	Calomel	... ...	gr.9¼
	Distilled water...	...	f ℥ivss
	Syrup acacia	... ...	f ℥ivss
	Acacia powder, q. s. to make emulsion.		

To be taken in one dose (shaken well) in the morning, patient having previously observed milk diet for twenty-four or thirty-six hours, and having remained in complete repose. Tænia expelled without any discomfort within thirty or thirty-five minutes.—*(Mercredi Médical.)*

A CLINICAL LECTURE

ON CASES OF

HEART DISEASE IN RELATION TO NERVE SYMPTOMS.

Delivered at the London Hospital, Nov. 6, 1894,

By FRANCIS WARNER, M.D., F.R.C.P.

Physician to the Hospital.

GENTLEMEN,—To-day I propose to speak on the subject of heart disease in relation to nerve symptoms. At the present time in Currie Ward there is a lad, 21 years of age, to whose case I will first call your attention. He had an attack of rheumatic fever last September, but except for that he is reported to have had no other illnesses. It is probable, however, that he may have been a rheumatic patient in much earlier life than the time of that illness. In the attack that he had last September there was pain in the region of the heart, but it does not seem to have been in any sense a severe attack, and we must conclude from the conditions found that the heart disease which I have to describe commenced long antecedent to the date of that illness. He has been quiet and intelligent, taking an interest in some clubs that he belongs to, and there is no reason whatever to think that he had any mental disturbance such as I shall have to speak of, on any former occasion.

On the 20th of September this year he was attacked with pain in the right leg; also in the chest, in the precordial region, and he became distinctly short of breath, in which particular he got worse until the 25th, when he sought admission to the hospital. On admission he was found to have great difficulty in breathing, and was decidedly anæmic. He complained that he had a considerable amount of head pain, and that sleep was difficult to obtain. On examination there was a distinctly collapsing pulse, which also was intermittent and irregular. I lay some stress on this alteration of rhythm, its being intermittent. There were distinct signs of a large left ventricle. The apex-beat was two inches outside the nipple line; the impulse was strong; there was a loud double

aortic murmur to be heard on the upper part of the right side of the sternum, and a systolic murmur at the apex, conducted towards the left axilla: in other words, he had all the ordinary physical signs of aortic regurgitation affecting the left ventricle. Some signs of circulation failing were found in the intermittency of the pulse. Then as a complication we found, although not to any very great extent, indications of pleuritic effusion on the left side. That does not appear to have played a very conspicuous part in the case, but still it indicated that there was some amount of congestion, but there was no œdema in any part. He presented no marked indications of disturbance of the nervous system, then, beyond the fact that he complained of some head pains and was very much distressed by want of sleep. The next note to which I need refer you is eleven days later, on October 5th. The patient during the intervening time had been sleeping very badly; there was a great deal of restlessness, and about the date I mentioned he became talkative at night and delirious. On several occasions he tried to get out of bed, and he had to be carefully watched. The next day there were distinct delusions, and he thought he saw the head of Christ in a blaze of light appearing through an aperture in the wall. He indicated that at the time by the things he said, referring to that which he saw. He thought that he was dead, and that he was going to his home. At another time he tried to get at a carving-knife, as he said to us then, "to cut up his young woman"; but in his normal state of mind he was an affectionate lad, and very fond of her. Then, among a third set of delusions, he imagined that a fire had been lighted under his bed, and he told us he was getting baked by it. You, therefore, see that there were various sets of delusions well established and clearly defined, such as we are quite accustomed to meet with in patients who are insane. That condition lasted for some days. It did not correspond to any definite increase in the bad action of the heart, and I may say that during a portion of the time he was able to sit up in bed, and when I saw him he had a newspaper in his hand. He looked at the newspaper, but he would not speak about it,

and there was no evidence whatever that he saw anything written in it. Still, as is the case with many patients who have delusions and who are insane, he sat for hours together with that newspaper in front of him, in a position as though he were reading it.

As regards the treatment in this case, I will not go into any details, but I may say that the one form of treatment which we found of use was hypodermics of morphine. In these cardiac cases, especially in aortic cases, when the patient is much distressed from insomnia I have no reason to regret the use of hypodermics of morphine up to $\frac{1}{6}$ of a grain.

Passing over all details, the next note I will refer to is on the 23rd October. By this time the patient had been enabled to sleep and was getting better. He was able to speak to us, answer questions intelligently, and said that he felt much better. He was conscious of some irregularity of the heart's action, and he now became more conscious of that irregularity than he had been when his mental condition was worse. By the 23rd October his speech was perfectly rational, and he could tell us what he was reading about. He remembered the delusions which he had had, and described them to us ; he was perfectly aware that they were delusions. Those are all the particulars that I need trouble you with concerning this case, except one other item, and that is about the eyes, which were myopic, and caused some difficulty in ophthalmoscopic examination. My colleague, Mr. Waren Tay, examined him for me. The only point I have to draw your attention to there is that we did not find a locomotor condition of the arteries of the retina. Of that I shall have to speak presently.

I give you the notes of this case briefly as an illustration that in heart cases we may meet with troublesome nervous symptoms.

To impress this further upon you I will refer to another case which I saw some years ago, and kept up with for several years. It was that of a girl about 12 years of age. The first time that I had to do with her in this hospital she was brought on account of chorea, from which she recovered. She had mitral regurgitation, but never had another attack of chorea. About a year after I was asked to see her again, as she had suddenly developed a marked tendency to steal. She went to a very good school in the neighbourhood, and it was found that she had been stealing right and left in a

very senseless manner from her friends, from home, from teachers, whenever there was a chance of her doing so. It was a regular attack of kleptomania. Happily for the child her friends consulted a doctor about her. Here was a child who had had one attack of brain disturbance and mitral regurgitation. She had previously suffered from an attack of chorea, which we know somehow or other is largely connected with heart disease. It seemed that the best thing for the child was to see what we could do towards recovering a healthy tone of brain and mind, by treating her as we had formerly done for chorea. She was kept in bed and put under high feeding. In the course of about three weeks the child had gained in weight. She was perfectly well, and had forgotten all her tendencies to steal. There was no further difficulty with her, and she was sent home, and returned to school. This would seem to be a case in which there was again a slight mental perversion, and that certainly was associated with disease of the heart. The girl is married and respectably settled in the country.

I may mention a third case, because it puts another aspect upon these symptoms, which may be associated with heart disease. Some years ago my colleague, Dr. Langdon-Down, had under his care, in this hospital, a woman having aortic and mitral disease. After she had been in the hospital some days she began gradually to take less and less notice of things around her, until she fell distinctly into the so-called cataleptic condition, and for about three weeks she remained in bed, showing no signs of consciousness whatever of any wants or of anything that was done for her. She was fed from time to time, the food being put into her mouth. She swallowed it, but there was no indication of any sort of response or consciousness. All through that time she had the ordinary dangers of aortic regurgitation, and I have no doubt that had not great care been taken with her it would have cost her her life. It may aid your memory of the case if I mention a little incident that occurred in connection with the patient. In some way a notice of the case had got into the columns of the daily papers. In consequence a large number of letters were sent to Dr. Langdon-Down on the conditions of catalepsy, mostly by women who had suffered from the condition themselves. One letter Dr. Langdon-Down brought with him, and read to his class at the bed-side of the patient. The lady who wrote the letter begged him to be careful what he

did, and on no account to allow the patient to be considered as dead, and buried. Her argument was that she herself some years ago had been in a cataleptic condition, in which she was supposed to be dead. A coffin was brought into her room, and her body was about to be placed in it, when she managed to give a little wink. Taking the hint, or following that, the patient we had here gave a little wink too, and in the course of a few days regained consciousness, and quickly recovered. You may say that this was only a case of hysteria. Be it so. I do not mind what name is given to it, but at any rate, if hysteria, it was so severe a case that the patient was in danger of her life. We may therefore have a cataleptic condition associated with conditions of heart disease.

Passing from these cases, I may speak of the general question, the relation of heart disease and nerve symptoms. It is well known that in many cases of hemiplegia there is valvular disease. If you watch the cases of heart disease in the hospital you will from time to time find such cases becoming hemiplegic; indeed, it is not at all uncommon to see such conditions occurring. As you know perfectly well, this may happen either through the occurrence of embolism, or through the effects of the heart in causing cerebral hæmorrhage. This is one class of cases of organic lesions of the nervous system following valvular disease. Then, as is well known, cases of chorea frequently present signs of mitral regurgitation. You must remember that, conversely, when you are dealing with a young child who has mitral disease, that one of the complications it is liable to is the onset of chorea. There are many other so-called neuroses, nerve conditions, which are found in varying degree to be associated with valvular disease. Epilepsy is one of these. If you look down any long series of cases collated to show the etiology of epilepsy you will find that valvular disease of the heart is included as one of the coincident conditions. Something like four or five per cent. of epileptic patients have heart disease. In taking a number of instances where heart cases have developed nerve disturbance, I find that the largest number of them are cases of aortic disease. In fact, you will find that a very large proportion, nearly one half, of the patients who have aortic disease suffer from such symptoms as insomnia—often a very difficult symptom to contend with—headache and vertigo. Many other nerve symptoms may occur in these patients also.

We must mention asthma, for a considerable proportion of asthmatic patients seem to have that neurosis associated with valvular lesions. When you have a case of a neurosis, it is important to look to the heart condition. I may mention the case of a lad who was attending one of the best public schools in London. He was brought to me for severe headaches, and I found that he had suffered a year before from a severe attack of influenza, after which these headaches had supervened. His father was a professional man, and it was intended that the son also should follow a professional career. The boy was fifteen years of age. So severe were his headaches, and so disabling were they, that it was considered necessary that he should give up all education, and on the advice of the head master, he had been taken away from school for a time. It appears that the headaches might be partly sequent to the influenza, and certainly in the sequelæ which followed influenza the headaches came first. At the time I saw him I found a distinct, loud aortic murmur. There was no doubt that the case was one of aortic regurgitation associated with development of headaches. In all cases of neurosis, therefore, it is very important to look to the cardiac condition. Before passing away from this branch of my subject, disease of the heart and nerve signs, let me remind you that there are also other cases of heart disease, not valvular, in which these nerve complications may occur. It is not at all uncommon to find all these nerve complications, of which I have spoken, coincident with hypertrophy of the left ventricle, without valvular disease, due to arterio-capillary fibrosis, so much associated with albuminuria and granular contracted kidneys. In the case of the late Czar of Russia, which has been attracting so much attention, the nerve symptoms attending on his undoubted cardiac hypertrophy, which subsequently failed, were very prominent and marked.

In further illustration, I will refer very briefly to a number of cases of heart disease, taken consecutively, with which we found nerve symptoms associated. In the list in front of me, I will not describe the form of heart disease (they are all cases of valvular disease of the heart), but I simply propose to pass in review the nerve complications attending it. They are mainly as follows: headaches, stammering, mania, epilepsy, chorea, convulsions, locomotor ataxy, tremors, asthma, and hemiplegias, and other forms of paralysis.

Now, among the list of conditions I have given here, I mentioned one of considerable interest, namely, stammering. Last week a little girl of 5 was brought to me from the country on account of her stammering and difficulty in speech. When she was between 3 and 4 years old this condition of stammering was noticed, and the parents were anxious about it; otherwise the child appeared perfectly healthy, well developed, bright, and intelligent. I went into the condition of stammering, and gave advice about it, and then I proceeded to make an ordinary physical examination of the child. Very much to my own surprise I found that there was a loud systolic bellows murmur localized over the third left cartilage, not well conducted, not attended with any other physical signs. There was no doubt whatever that it was a case of congenital defect of the heart. Now, this made a great deal of difference to my view of the case, in the prognosis that had to be given; not only is there the possibility of defects in circulation following from that defect, but also it altered my view of the condition of the brain causing stammering in that case. Where you have a nerve defect in a child, and you find it to be concurrent with some bodily deformity or congenital defect in its development, you have much more reason to look upon the nervous condition as of grave importance. When I found that the stammering was associated with the defective heart, I felt that the chances of the child's getting rid of it were less favourable than had it been in a child who presented no other defect whatever of any kind except the phenomenon of the stammer. Similar conditions are found in other cases in which stammering has been the principal condition noted.

This, then, takes me to another division of my subject — the presence of congenital defects of heart in association with nerve symptoms. In imbeciles it is not at all uncommon to find that along with the defects of brain and various defects of body they have also congenital defect of the heart, which of course affects the question of prognosis. I will refer you to a few cases which have come under my notice at various times. A boy, 3 years of age, came under my care, who presented indications of defect of the heart. The apex was displaced, and there was a loud systolic murmur of varying intensity heard at the base, no clubbing of the fingers, and no cyanosis. This case was one which presented other deformities also; the hands were deformed. It occurred in a family in which there had been three cases of epilepsy. The fact that this boy had congenital defects in the make of his body and in the make of his heart, with a family history of a strong predisposition to epilepsy, made us fear very much that he might develop epilepsy : for where there is a defect of the heart, nerve disturbances are much more likely to occur.

Then as regards the association of nerve signs with congenital defects, I might refer also to the experience of asylums where imbeciles are collected—that is to say, children with congenital defects. It is found that a much larger proportion of the children in the epileptic wards, where imbecile epileptics are accumulated, have defects of the heart than among imbeciles generally; in other words, the association of heart disease with brain disturbance is very marked in all these cases of congenital defect, as well as it is in cases where the heart lesion has been acquired during the course of the patient's life. I have thus far spoken mainly of the nerve symptoms as empirically associated with the heart lesion. It seems, however, quite possible that there is a causal connection between the two. In cases of aortic regurgitation, if you look at the brachial or the radial artery as seen under the skin of the forearm, you may see a condition termed locomotor. The artery is moved by the force of the ventricular systole, and moves in locomotor or a snake-like fashion under the skin. An irregular supply of blood is the result. An effect of this kind of impulse passing through the arterial system is noticed when you look at the fingers. In some of those cases you will find that the nails are thin, and looking at the course of the circulation in the matrix under the nail, you may see the colour flashing with each systole of the heart, showing that there is a capillary ebb and flow instead of that uniform continuous vascular supply in the capillaries, which is the normal, and which we have every reason to believe is very necessary for healthy nutrition, particularly in the conditions of the circulation of the nerve centres. There is another way in which this irregular capillary supply in aortic cases may be observed. A very common way of observing the capillary circulation is to look at the lips of the patient. You take a microscope slide, and gently laying it on the mucous membrane of the lip, you can see the ebb and flow of the capillary circulation in the mucous

membrane. In some cases, also, it may be seen in the arteries of the retina; in aortic cases, the locomotor condition of those arteries is often clearly perceptible; the arteries appear almost to jump from their beds at each systole of the left ventricle.

It appears probable, then, that in at any rate many of our cases of valvular disease, especially those of aortic regurgitation, we have an irregular instead of continuous blood supply to the capillaries of the cerebral tissue. I think that the large association which we find between valvular disease and the development of nerve signs in such cases is probably due, in part, at any rate, to the presence of this irregular capillary circulation.

The general bearing of the remarks that I have made would seem to show that in any case where we are dealing with some nerve or neurotic condition it is exceedingly important to examine the heart, and not only to examine the heart sounds and impulse, but also to try and estimate the conditions of circulation, that is, the supply of blood as traversing the arteries, and the supply of blood as distributed to the capillaries of the tissues. But, of course, the circulation in the capillaries supplying the nervous system depends not only upon the course of the general circulation, but also upon the condition of the blood which is circulating in it. Watching the effects of anæmia, and other altered conditions of the blood, upon the condition of the brain, strengthens the ground upon which I entertained the views which I have given you. We often find anæmic patients develop various neuroses, though the course of the circulation—the pumping—is good. Very common is it to find not so much insomnia as an excessive condition of somnolence. Headaches are very common. Neuralgias, both in the branches of the trigeminal nerve and in the intercostal nerves are also very common where the blood supplied to the tissues is poor and out of order. Then, again, we get evidence that nerve disturbance may result from altered conditions of the circulation, in the fact that when the blood is of abnormal temperature, as in fevers, we also get a large amount of nerve disturbance. Among the early signs of fever, of pyrexia, we have pains—nerve pains, head pains, and pains in various parts of the body. Then we have weakening of the mental power, and very often a distinct amount of disturbance of the mental power which may pass on to the condition of active delirium and mental disturbance, leading almost to the conditions allied to mania. We see then that neurosis may occur as the result of a defective condition in the mechanical supply of the blood, and also in the conditions of the blood itself. As to these associations of anæmia with neurosis, there is no doubt that another element which enters into the case is that not only is the blood as supplied to the portions of the brain defective in its constitution, but also the patient is liable to very great irregularity in heart action. In anæmia the patient suffers very much from palpitation; the rapidity and frequency of the heart varies much under very slight stimulus, such as any cause of mental excitement. This in itself causes the varying condition of mechanical supply of blood, as well as leaving the blood itself in very poor condition.

Now, as to the treatment in cases in which these two sets of conditions are present—disturbance of heart and nerve cases. The remarks that I have made show the importance of considering the condition of circulation in the treatment of neurosis. I will refer to a few cases in which the matter assumes a particularly practical form. For example, in headaches you have to treat a case of megrim. One of the most important things to do is to bring about the best condition you can of cerebral circulation; hence one of the advantages of making the patient lie down during the attack. Again, in an attack of megrim you very often find that the feet are very cold, and getting the feet warmer restores a better condition of circulation, indicated by the pulse, which itself leads to a better condition of the action of the brain of the patient. The same point may be illustrated sometimes in fever cases. A man presented himself last week at the out-patient department, with a facial expression of great anxiety, very marked in the muscular action of the forehead, and at the same time he was in a condition of general tremor, showing the great weakness of his nervous system. The patient's temperature at that time was only 101°. I at once made a diagnosis, and concluded that the patient was suffering from typhoid fever. We took him in, put him to bed, and in a very short time the look of anxiety and the condition of tremor had passed away completely. Here was a case where the special neurosis to which I refer, the facial aspect and the tremor, were at once relieved by putting the patient to bed, keeping the body at a uniform temperature all over, and so giving a more uniform condition of circulation.

The same principle will aid you in treating a good many other cases. Take, for instance, cases of chorea. Many cases of chorea which continuously get worse while the patient is going about, will improve in a marked degree when put to bed, causing (even though there may be mitral regurgitation) a better condition of the general circulation. The same thing also applies to the use of drugs in neurosis. As a marked example of this I would refer to a case of delirium tremens. In delirium tremens the great thing is to get rid of the delirium, and experience shows that the best modes of treatment are those which serve to strengthen the heart and circulation. For this reason it is very important to keep every source of excitement away which not only acts upon the patient's brain but increases the heart's frequency. Heart tonics prove of the greatest use. Digitalis is often most useful in removing delirium, and alcohol also, for the same reason, is necessary in some cases of delirium tremens and other forms of delirium. It probably acts by no means simply upon the brain directly itself, but improves the brain condition by improving the conditions of circulation. In this you find one of the criteria by which you may get an indication as to whether stimulants are called for. Delirious cases, where circulation is failing (indicated by the pulse, the heart sounds, and impulse of the heart), are improved by the administration of stimulants. If you bear these remarks in mind you will find that they are of considerable importance when looking at the condition of the nervous system. In all cases of heart disease examine carefully the state of the nerve system; and equally important is it in all cases of nervous disturbance to examine and treat any co-existing defects in the heart and the circulation.

Lotion for Conjunctivitis.—

R   Acidi borici   ...   ...   gr.xx
     Sodii chlorid. ...   ...   gr.viij
     Aq. dest.   ...   ...   ℥ij

M. Ft. lotio. Use freely every four hours, first warming.   (*Therapeutic Gazette.*)

For Small Wounds and Abrasions.—

R   Iodol ...   .   1 part
     Ether ...   .   10 parts
     Gun cotton   ...   ...   5 parts

Apply locally.   (HARE, *Coll. and Clin. Rec.*)

A POST-GRADUATE LECTURE

ON

BILIARY CALCULI.

Delivered at the Metropolitan Hospital by

H. J. WARING, M.S. Lond., F.R.C.S.,

Assistant-Surgeon to the Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital.

Definition. Gall-stones, biliary concretions, or biliary calculi, are solid bodies of varying size and shape, which have been formed as a result of pathological processes occurring in some part or parts of the ducts of the liver.

Mode of Formation and Pathology. In the majority, if not in all cases, the formation of these concretions is associated with the presence of an obstruction to the free outflow of the bile from the biliary duct into the duodenum. The mere existence of an impeded biliary flow, however, does not of itself seem to be sufficient. The exciting cause appears, as has been shown by Naunyn, to be an inflammatory condition of the mucous membrane lining the biliary system, which has been set up by the presence of a micro-organism known as the "bacillus coli communis." This bacillus is a constant inhabitant of the alimentary canal, and when the flow of bile along the common bile duct into the duodenum has been slowed, it obtains an entrance into the biliary system by passing along the common bile duct, thence into the cystic duct and gall-bladder, and also into the hepatic ducts. After obtaining an entrance it multiplies, acts as an irritant, probably owing to the chemical products known as "ptomaïnes," and in this way sets up an inflammatory process in the epithelial cells on the internal surface of the mucous membrane. As a result of this inflammation, the metabolic processes of the epithelial cells appear to undergo considerable modification. If the cells are examined with the microscope, many will be seen to be swollen, and their interior to be occupied by myelin-like masses, which are being extruded upon the surface of the mucous membrane, and there crystallising to form small masses of cholesterin. In some places several of these swollen cells will be seen to be aggregated together forming a small mass in which there may be a few blood corpuscles. On the surface of this mass crystals of cholesterin are deposited, and thus the formation of a biliary

concretion is commenced. Occasionally a foreign body, such as a pin, has been found in the centre of a gall-stone, or the dried-up remains of an ascaris (or round-worm), or of a distoma (or fluke), or of a small hydatid cyst. In some cases the core is composed of calcium carbonate crystals which have been formed as an excretion from the epithelial cells lining the gall-bladder. In other cases, again, small casts of the intra-hepatic bile capillaries seem to have been the nucleus around which a deposit has taken place with the resulting formation of a calculus. Small plugs of inspissated mucus also appear to form the starting-point of the deposit.

Place of Formation. In nearly all cases gall-stones are to be found in either the gall-bladder, or the cystic or common bile duct, much more rarely in some part of the right or left hepatic ducts, or their smaller ramifications. Occasionally a stone is found in the common hepatic duct, having passed backwards from the gall-bladder. The gall-bladder itself is the place where nearly all gall-stones have their origin ; but possibly a few commence in the commencement of the intra-hepatic bile capillaries.

Structure. A gall-stone generally consists of three portions : (1) a central core or nucleus ; (2) a middle layer of considerable thickness, consisting of radiating crystals of cholesterin; (3) an external, thin, dense laminated layer of bile pigment and cholesterin.

The composition of the nucleus has been discussed above. It generally has a dark green or somewhat black appearance, owing to the presence of pigments. In some nuclei numerous bacilli have been found. Usually it is solid ; but occasionally, owing to dessication, a honeycombed appearance has been seen. The middle layer is usually the thickest, and consists of radiating crystals of cholesterin, which have a laminated arrangement. This layer may be white or light-coloured in appearance, but often is somewhat yellow, owing to the permeation of a certain quantity of the bile-colouring matter. In a few cases this middle layer has a soapy appearance, and shows no sign of laminæ or crystallization.

The external layer is thin, dense, and dark green or brown in colour. It has a varied consistence. Sometimes it is composed of layers of cholesterin or of calcium carbonate, alternating with layers of pigment, or it is made up of a compound of calcium carbonate and bile-colouring matter. In a few cases this external layer is absent ; the surface of the stone is then rough, and is formed by the free ends of the radiating striæ of cholesterin, upon which has been deposited a small amount of pigment.

Varieties. The intra-hepatic calculi in nearly all cases consist of a compound of calcium and bile pigment, but occasionally they consist of pure cholesterin. Those which take origin in the gall-bladder have been classified by Naunyn as follows :—

(1) Calculi composed of pure cholesterin.

(2) Stratified cholesterin calculi.

(3) Common biliary calculi, which are usually multiple, numerous, and facetted.

(4) Mixed bilirubin calcium calculi, which are single or few in number, and when multiple may be facetted.

(5) Calculi of pure calcium bilirubin, small and rare.

(6) Rarer forms of biliary calculi, in which are included (*a*) small, amorphous, or imperfectly crystallized cholesterin calculi ; (*b*) calculi containing calcium carbonate ; (*c*) conglomerated calculi ; (*d*) casts of the intra-hepatic ducts.

Occurrence. (*a*) Sex. In the majority of cases biliary calculi are found to occur in the female sex. Schroeder, in an investigation carried on at Strasburg, found gall-stones in 4.4 per cent. of all male subjects, and 20.6 per cent. of all female subjects examined.

(*b*) Period of life at which they occur. Under the age of 30, biliary calculi are very rare. After that age they are much more frequent ; between 30 and 60, they are found in over 10 per cent. of autopsies ; whilst over 60, they were found in one body out of every four examined.

Causes. Predisposing causes. All those conditions which impede or obstruct the flow of bile along the biliary passages favour the formation of calculi. The movements of respiration, especially of the abdominal type, are important factors which help the biliary flow; hence, when any derangements of this process occur, the possibility of the formation of biliary calculi is increased. In pregnancy, for example, the free descent of the liver is impeded, and the intra-abdominal pressure is increased, while often the anatomical relations of the abdominal viscera are altered. An intra-abdominal tumour, such as cystic disease of the ovary, kidneys, etc., acts in a similar manner. The tendency to the formation of biliary calculi

in women is also often increased by tight-lacing and sedentary habits.

Diseases of the intra-thoracic viscera which tend to impede the free movements of respiration, often are predisposing causes. Age, also, is an important factor. Charcot has shown that the muscular tissue in the walls of the gall-bladder and biliary ducts undergoes considerable atrophy in old age. On this account there may be much less likelihood of a calculus being expelled in old age than in the early periods of life. These observations help to explain the frequency with which biliary calculi are found in the gall-bladder after death, and also to the comparative rareness of attacks of biliary colic in old people in whom, after death, concretions are found to be present. The existence of ascarides or distomæ in the alimentary canal or hepatic system increases the probability of the formation of gall-stones.

Cancer of the gall-bladder or bile-ducts has, by some authors, been said to be an important predisposing cause. Usually, however, the calculi have been present before the onset of the malignant disease; but it is quite possible, and even probable, that in a few cases the cancerous growth is present and is followed by the formation of biliary calculi.

Symptomatology. (a) Biliary calculi may exist in the gall-bladder, or occasionally in some part of the bile-ducts, without giving rise to any symptoms during life by which their presence can be diagnosed. Usually the first symptoms noticed are pains in the upper and right side of the abdomen, in the right hypochondriac and epigastric regions. These pains may be only slight and constant, giving rise to a feeling of fulness and heaviness in the right hypochondrium, together with a dragging sensation. Usually, however, there occur, sooner or later, paroxysms of severe pain. This is localised in the right hypochondriac or epigastric region, from which it radiates forwards over the abdomen towards the umbilicus, and backwards towards the inferior angle of the scapula. These paroxysms may come on after some variety of exercise, or after taking food; but often they come on spontaneously without apparent exciting cause. Usually they occur in the daytime, when the patient is up and about, but occasionally a patient will wake up in the night with a paroxysm. The attack may last for a few minutes or hours; it is usually of several hours duration. Often the pains in a paroxysm are very severe, and, if pro-

longed, may give rise to collapse, sickness, or vomiting. Frequently, when an attack has lasted a short time, a feeling of sickness followed by vomiting occurs, and then the attack comes to an end. The paroxysms of pain, which are very often called "gall-stone colic," may be either single or repeated; and are excited by the passage of a calculus from the gall-bladder into the cystic duct, and thence outwards into the duodenum through the common bile duct. Occasionally the stone may be arrested at some point in its passage, and according to the site of the point of arrest, there will follow a train of symptoms which will be described later.

(b) *Sickness.* A feeling of sickness and general malaise may occur which is quite independent of the paroxysmal attack. It will usually be associated with the constant hypochondriac pains, and often is dependent upon the irritation of the calculi in the gall-bladder, or when a calculus passes into the neck of the gall-bladder but is too large to pass along the duct. This symptom is more common in old people who suffer from biliary calculi, and in whom the muscular tissue in the walls of the biliary system has undergone a certain amount of atrophy and degeneration. Often an intense feeling of nausea precedes an attack of vomiting.

(c) *Vomiting.* Towards the end of a paroxysm an attack of vomiting often occurs. The vomited matters are, first, the contents of the stomach, and then bile-stained material, and, in rare cases, a biliary calculus will be found. In some rarer cases, when the vomiting is persistent, the vomited matter may have a stercoraceous odour.

(d) *Collapse.* If a paroxysm is protracted in duration, or the fit of vomiting severe, collapse, more or less marked, is the result. A certain amount of collapse is the usual result of a paroxysm, the patient being then weak and prostrated, with a small feeble pulse, a cold clammy skin, and a lowered temperature. Mayo Robson has recorded a case in which a patient died from collapse produced by a protracted and severe paroxysm. After death a calculus was found half-expelled from the common bile duct into the duodenum, and still grasped by the ring of muscular tissue in the terminal part of the wall of the common bile duct.

(e) *Presence of Calculi in the Fæces.* If the stools are examined after a paroxysmal attack, there will, in some cases, be found smaller or

larger calculi in the excreta passed *per anum*. To find the calculi the excreta should be mixed with a weak solution of carbolic acid in water, then well mixed up, and finally passed through a fine wire sieve of which the meshes are about 1–12th in. square. When found, the structure and composition of the calculi should be examined, so as to make certain of their nature. In some cases, after a paroxysmal attack no calculus at all will be found. In these cases the calculi have either become lodged and arrested in some part of the biliary system, or in some part of the intestinal canal, or the calculus may have slipped back into the gall-bladder.

(f) Jaundice. When a calculus becomes impacted in the common bile duct there will be produced an obstruction to the flow of bile from the liver into the alimentary canal, which, if nearly complete, gives rise to an attack of jaundice. The conjunctivæ are stained yellow, and afterwards the skin, and the urine becomes deep coloured, owing to the presence of bile pigment. In the majority of cases an attack of jaundice, when it occurs in connection with calculus, continues a short time after the paroxysm of pain—generally a few days afterwards. In rare cases attacks of jaundice may occur in connection with and as a result of biliary calculi without any attacks of pain. The usual course of events, however, is an attack of severe pain or biliary colic, followed by jaundice more or less intense, and dependent upon the completeness of the obstruction. In these cases the calculus becomes arrested in some part of its course from its seat of formation to the duodenum, and blocks up the lumen of the bile canal. The bile secreted by the liver collects behind the obstruction, and, when the pressure has risen, it passes into the lymphatics of the liver and is carried into the thoracic duct, and thence into the general blood circulation, whence it is partially deposited in the skin and conjunctivæ, and partially excreted by the kidneys in the urine. The fæces become clay-coloured, of foul odour, and constipation is present. If the obstruction is only partial the fæces may still retain their normal colour. This sequence of events—namely, paroxysmal attacks of hepatic colic, followed by jaundice with its accompanying symptoms—is very diagnostic of the presence of biliary calculi.

(g) Occasional high temperature. In many cases of biliary colic associated with jaundice there is present as well an ague-like temperature, which

in many respects resembles an attack of urethral fever. Soon after the termination of an attack of colic the patient has a rigor, the temperature rises to 103° F. or more, the rigor lasting for a variable time, but generally of short duration. This rigor is then followed by a fall of temperature and some prostration, from which the patient soon recovers, only, however, to suffer from successive similar attacks. These rigors may occur without any jaundice, but they are much more usual when jaundice is present. As regards their pathology little is known. Some authors (such as Charcot) have maintained that the fever was due to the absorption of some ferment which had been produced in the bile passages, whilst Murchison and Ord say that it is nervous in character, and is due to irritation of the mucous membrane of the common bile duct by the calculus.

When a calculus becomes fixed in the common bile duct it is generally arrested just above the orifice in the duodenum, in the small dilatation known as the ampulla of Vater. Two conditions may arise as the result of the arrest of the calculus. (1) A catarrhal condition of the biliary passages, which has fairly defined symptoms, with ague-like attacks, rigors, fever, etc., is intermittent in character, and often long persistent; (2) biliary obstruction associated with a suppurative process in the biliary ducts. When this occurs the fever is generally of a remittent character.

(h) If the hepatic region of the patient be examined, the liver in some cases may be found to be tender to palpation, and slightly enlarged, so as to project below the right chondral margin; but much more commonly a localized enlargement may be felt behind the ninth or tenth costal cartilage, which tends to extend downwards and forwards towards the umbilicus or along a line drawn from the ninth costal cartilage to a point one-third of the way distant between the umbilicus and the anterior superior iliac spine of the right side. This swelling is smooth and rounded, larger below than above, moves with respiration, descending during each inspiration and ascending during each expiration, and can also be moved laterally by pressure with the examining finger; it is dull to percussion and dumb to auscultation.

In order to palpate the abdomen, the patient should be directed to lie upon his back, and with the knees slightly flexed. Then he should be told to breathe quietly, and the palpating hand should be laid flat upon the abdominal wall over the hepatic

region, and pressure gradually increased, especially at the tips of the fingers. In this way, if there is any abnormal enlargement in the hepatic region, it can, as a rule, be felt. If, however, nothing can be made out in this way, the patient may be directed to sit up and to bend the body slightly forward, breathing as before. The hand should then be applied to the hepatic region, and pressure gradually exerted with the fingers. In this way it is often possible to palpate swellings in the region of the gall-bladder which cannot be made out otherwise. It is possible in many patients, especially women with thin and lax abdominal walls, to feel the normal notch in the anterior inferior border of the liver, in which lies the fundus of the gall-bladder.

If both those methods fail to detect anything, it will then be advisable to anæsthetise the patient. If a swelling is felt in the region of the gall-bladder, or having its point of attachment in that region, it will usually have the characters already mentioned, and in addition there may or may not be a feeling of fluctuation. This fluctuation is difficult to detect, owing to the tenseness of the dilated gall-bladder. If, however, the tumour consists chiefly of calculi, no fluctuation will be obtained.

(i) Occasionally, without the usual symptoms of biliary colic, the impaction of a large gall-stone, which has passed into the alimentary canal, may by ulceration cause acute intestinal obstruction. This must be remembered in considering any case of intestinal obstruction of which the cause is not clear, and the region of the gall-bladder should be examined in order to find out if there are signs to be felt there which may be of use in making a diagnosis.

Diagnosis. The most important symptoms which point to the existence of biliary calculi are the paroxysmal attacks of lancinating pain with a feeling of sickness, and terminating usually in a fit of vomiting. These pains are most marked in the hypochondriac region, radiating from there down the abdomen towards the umbilicus, and backwards towards the inferior angle of the right scapula. Jaundice, when present, usually follows the painful attacks, is variable in intensity, and generally transient in nature. If it is persistent, and lasts for months, the existence of carcinoma, involving some part of the liver or biliary system, is to be suspected. If calculi are vomited or passed *per anum* the diagnosis becomes certain.

Collapse and exhaustion are symptoms which follow the attacks of pain and vomiting, and, if severe, usually indicate the presence of biliary calculi. Biliary colic can usually be distinguished from renal colic, due to the passage of a calculus along the right ureter, by the fact that the pain radiates downwards along the right ureter to the pelvis, and is associated with symptoms of vesical mischief and pathological conditions of the urine. In the colic of lead poisoning there will be chronic constipation and a blue line on the gums, whilst the symptoms soon disappear under appropriate treatment. In gastric affections and dyspepsia the pain is most intense on the left side of the abdomen, and is accentuated by food. In cancer of the head of the pancreas, which involves the common bile duct, a tumour will usually be felt in the posterior part of the abdominal cavity in the region of the pancreas, which is fixed, and does not move on respiration. Persistent jaundice will also be present. Persistent jaundice, not associated with periodic attacks of pain or rise of temperature, especially if rapid loss of flesh is also present, will point to malignant disease of the biliary ducts or gall-bladder or liver.

Membranous enteritis may produce symptoms very similar to those of biliary calculi. If the fæces be examined casts or flakes of the false membrane may be discovered, which will help to discriminate between the two affections.

In abscess of the liver the pains are not so severe, and do not occur in paroxysms or radiate over the abdomen so extensively as in biliary colic.

The passage of an ascaris (or lumbricoid worm) from the duodenum into the common bile duct, will give rise to symptoms exactly similar to a severe attack of biliary colic, and cannot usually be distinguished from it; but it will probably be in a young person in whom gall-stones are not common, and the person affected may give a history of having passed these worms *per rectum*, or *per oram* by vomiting.

The presence of a hydatid tumour in the liver which bursts into the hepatic or common bile duct, and the passage along this of the daughter cysts, will give rise to paroxysms of pain similar to those due to the passage of a calculus. They will be distinguished by the previous existence of a fluctuating hepatic tumour which has suddenly diminished in size, and by the presence in the fæces of hooklets and portions of the membrane which formed the wall of the cyst.

In order to distinguish a tumour formed in

connection with the gall-bladder from cases of renal, supra-renal, or ovarian tumour, it may be requisite to apply Ziemssen's test. This is carried out by injecting a quantity of air *per anum*, sufficient to dilate the rectum, sigmoid flexure and colon, and then percussing the region of the tumour. If the tumour is pushed upwards towards the thorax, and the marked resonance is below it, then, in all probability, it is either hepatic or in connection with the gall-bladder; whilst, if it is pushed downwards and backwards, and the marked resonance is above, it in all probability will have no connection with the liver or biliary system. Some surgeons have advised puncture with a small trocar or an aspirating needle in order to determine the nature of the tumour in the region of the gall-bladder. This proceeding, in my opinion, is rarely justifiable, since the puncture is liable to leak afterwards and the contents of the swelling, if fluid, to pass into the peritoneal cavity, and set up a peritonitis which may prove fatal. Sounding with a needle is, I consider, a most barbarous method, and in my opinion ought never to be resorted to. If it is requisite to explore, in order to make a correct diagnosis and to clear up an obscure case, the best method is to make a small median incision or an incision in the right linea semilunaris, just below the ninth costal cartilage, through the anterior abdominal wall above the umbilicus, and large enough to admit the finger, and through this to make a thorough digital examination. This can be done with practically no risk to the patient, whilst the surgeon is not working in the dark, as is the case with a long exploring needle.

Complications and Sequelæ. The presence of biliary calculi in the excretory ducts give rise to symptoms which may be used to artificially divide the cases into two main groups. The first group includes all those cases in which calculi are present in the gall-bladder, and in which periodical attacks of pain occur owing to the passage of a stone from the gall-bladder into the duodenum. The second main group includes the remaining cases. It includes :—

A. Cases in which impaction of a calculus takes place in some part of its passage from the gall-bladder and duodenum. This impaction may be in :—

(1) The neck of the gall-bladder.

(2) Some part of the cystic duct.

(3) The common bile duct, usually in the ampulla of Vater.

(4) The hepatic duct, when the calculus passes backwards towards the liver.

The impaction may be such as to produce either partial or complete blocking of the flow of fluid along the duct in which it is impacted. If the cystic duct is the seat of impaction and the obstruction is complete, usually the gall-bladder becomes dilated owing to the accumulation of mucus which is secreted by the glands in the membrane lining the internal surface of the gall-bladder. In this way the gall-bladder may attain enormous dimensions, filling up a considerable part of the abdominal cavity. Usually, however, it does not extend beyond the umbilicus. If the calculus is impacted in the common, hepatic, or bile duct and the obstruction is complete, there will be accumulation of bile in the portion of the duct proximal to the obstruction, followed by jaundice. If, however, the obstruction is in the common bile duct, and, owing to a previous process of ulceration, by means of which a calculus has passed from the gall-bladder or cystic duct directly into some part of the alimentary canal, a fistula has been left, then there may be no symptoms beyond the irritation caused by the presence of the stone. The gall-bladder usually does not become dilated in cases of obstruction of the common bile duct, owing to the presence of a spiral valve in the interior of the cystic duct which prevents the easy passage of fluid towards the gall-bladder.

B. Cases in which impaction of the calculus occurs, and is followed by an infective suppurative inflammation of the mucous membrane lining the biliary passages. As a result of this, we may have produced :—

(1) Inflammation of the bile ducts and gall-bladder.

(2) Empyema of the gall-bladder.

(3) Hepatic abscess.

(4) Abscesses in structures in the immediate neighbourhood.

(5) Acute suppurative general peritonitis.

C. Ulceration of the walls of the gall-bladder or bile duct, followed by adhesion to a neighbouring portion of the alimentary canal — duodenum, colon, or small intestine—and perforation into the lumen of the gut, with passage of the calculus into the alimentary canal.

D. Intestinal obstruction owing to the impaction of a gall-stone in the alimentary canal, which may be due to (1) passage into the duodenum through

the common bile duct; (2) ulceration and perforation.

E. Adhesion of an inflamed gall-bladder to the posterior surface of the anterior abdominal wall, followed by the formation of an abscess which points on the surface and may burst; discharging pus and gall-stones, leaving a biliary fistula.

F. Inflammation of the gall-bladder, followed by dessication of the pus, and conversion of the gall-bladder into a mass of fibrous tissue, or a calcareous mass.

G. Ulceration of the mucous membrane lining some part of the biliary passage, followed by cicatrization and stricture of the lumen of the duct. As a result we may have all the signs of obstruction produced by a stone.

H. The continued irritation of the mucous membrane by calculi, may give rise to carcinoma (a) of the gall-bladder; (b) of the bile ducts.

Treatment. The treatment of biliary calculi has to be considered under two distinct headings :—

(1) Medical and general therapeutical measures.

(2) Operative procedures.

If a patient is suffering from an attack of biliary colic, placing the sufferer in a hot bath, or the application of hot fomentations to the hepatic region may afford temporary benefit, and also, by relaxing the tissues, may facilitate the passage of the calculus onwards into the duodenum. In some cases an injection of ¼ gr. of hydrochlorate of morphia under the skin will afford considerable relief. It may be requisite to repeat this dose. If the paroxysm is very severe, inhalation of chloroform should be tried. When the paroxysm has passed off, rest in bed should be combined with the application of warmth and the administration of some stimulant, such as brandy, ether, or strychnine, in order to restore the patient from the collapse and exhaustion which follow the attack.

The removal, absorption, and solution of gall-stones which are already present appears to be almost impossible by therapeutic measures. Some authorities advise belladonna and podophyllin, others olive oil, given by the mouth, as remedies which will cure gall-stones. There is little, if any evidence, however, of the value of these remedies. If there are only a few small calculi present in the gall-bladder they may possibly be passed spontaneously or with the aid of the above-mentioned drugs; and the formation of others may be prevented by attention to diet, exercise and the general hygienic condition of the patient. A moderate amount of

alcohol only should be allowed, whilst all indigestible foods should be forbidden. The bowels should be kept freely open by the administration of alkalies and aloes; Carlsbad salts, or sodium sulphate should be used for this purpose. A fair amount of gentle exercise should also be enforced, and if the patient be a woman, attention should be paid to tight-fitting corsets or any form of dress which would press upon the hepatic region, and impede the free movements of respiration.

Massage of the abdomen has been recommended as a measure which facilitates the expulsion of calculi from the gall-bladder or biliary ducts. This is not to be recommended, owing to its uncertainty and to the fact that ulceration or perforation of the walls of the bladder or duct may be, and has been, induced. All the above, however, can only be regarded as either palliative or preventative measures; and in order to cure the patient of the diseased condition, recourse must be had to direct surgical operative interference.

The signs and symptoms which point to the necessity of surgical interference are :—

1. The presence of a tumour in the abdomen which appears to be a large and distended gall-bladder.

2. Jaundice, which is persistent, together with other signs and symptoms which point to complete obstruction of the common hepatic or common bile duct.

3. The occurrence of successive paroxysmal attacks of biliary colic, with short intervals between, which are lowering the general health of the patient, and inducing a state of general exhaustion, and are not amenable to medical measures.

4. Symptoms of localized inflammation in the region of the gall-bladder, associated with attacks of biliary colic.

5. The occurrence of acute peritonitis, probably the result of perforation of the gall-bladder or bile ducts.

Prognosis. The prognosis in cases of biliary calculi is generally good. During recent years the advances made in the surgical treatment of diseases of the liver and biliary system have lessened the mortality from the various complications which are liable to arise as the result of the presence of biliary calculi. In inflammatory cases in which there is perforation into the peritoneal cavity, the prognosis is the worst; but even here, if radical surgical treatment is adopted immediately, there is a fair chance of the patient's recovery.

CLINICAL CASES WITH COMMENTS.

Read before the Folkestone Medical Society, Nov. 28, 1894,

BY

W. JOSEPH TYSON, M.D., M.R.C.P. Lond.,

Senior Medical Officer, Victoria Hospital, Folkestone.

(Concluded from page 180.)

Two Cases of Chronic Osteitis.

Case 1.—Osteitis of the inner malleolus of left tibia.

George S., aged 26, admitted into Victoria Hospital, March 22nd, 1893. The trouble began twelve years ago, after a sprained ankle. The latter ever since has been more or less painful. Six weeks ago the skin over the bone was divided on account of the increase of swelling. The condition on admission showed considerable swelling about the internal malleolus; the latter is very much thickened with periostitis and osteitis. There is a sinus at the posterior part of the malleolus, on probing which bone can be felt. Under an anæsthetic the periosteum was thoroughly divided. No sequestrum was found. The wound was well irrigated, dressed with iodoform emulsion and Lister's cyanide gauze. The wound soon healed, and the pain disappeared. I heard of this man a few days ago, and he is now quite well.

Case 2.—Osteitis of the shaft of the right tibia.

George P., aged 24, admitted into Victoria Hospital, May 5th, 1893. There is no history of tuberculosis. Thirteen years ago the patient had a kick on the foot, and some necrosis of one of the toes followed.

Present illness. States that 3½ months ago he had a blow on the tibia from the fall of a stone; a swelling then began to form in the bone, but only became painful nine weeks ago.

On admission. There is a large lump over the front of the middle third of the tibia, the size of a small orange, slightly painful on pressure, and becomes much more painful at night; it is very hard; there is no inflammatory redness. Temperature, normal.

On the 9th of May the periosteum was incised over the whole extent of the tumour, the bone trephined, and the edges of the cavity chiselled away. The wound was then irrigated and packed with gauze. Since the operation all pain has ceased, and the man has returned to his work. He is at the present time well and free from all pain.

Remarks. It is worth noting that such cases as that above described rarely get well—that is, they seldom get rid of the pain until the periosteum is thoroughly divided by linear osteostomy (in some cases, as in my second one, it is deemed advisable to remove a good-sized portion of bone as well). Of course one is not speaking of rheumatic or syphilitic nodes—these being both relieved by iodide of potassium.

The next two cases I will shortly mention are those in which the middle ear was diseased, the mastoid antrum at the same time being involved.

Case 1.—Frederick M., aged 21 months, was admitted on January 9th, 1893, with an offensive purulent discharge from the left ear. The discharge had existed for two months. A week before admission facial paralysis supervened. There was considerable swelling over the mastoid.

On January 12th the mastoid cells were laid open by means of a chisel, and a communication established between them and the middle ear; the granulations in the middle ear were scraped out, and a plug of cyanide gauze soaked in iodoform emulsion was put in. The child did well as regards the acute symptoms, although I am sorry to say that a sinus still remains. At present the parents refuse any further treatment, but it would be wise to remove any irritating materials which doubtless at present exist and account for the non-closing of the wound.

Case 2.—Albert H., aged 7 years, admitted into the hospital on November 10, 1893.

A purulent and offensive discharge had existed for one year, and for the week before admission there had been tenderness and redness over the left mastoid process. On the same day the mastoid antrum was opened; a thick piece of silver wire was passed from the antrum into the middle ear, and brought out at the external meatus, and then bent in the shape of a ring and allowed to remain in. The granulations in the middle ear were curetted. The wound was dressed as in the former case. The wire was moved every day; this was continued for a fortnight. The child is now cured, and hears fairly well on this side.

Remarks. The importance of the antrum of the mastoid process has only been recognised the last few years. When I was a student its existence was hardly known, and except that now and then, its periosteum was divided, it was left surgically at peace.

The anatomical position of the antrum is in such close relationship with the middle ear that it frequently becomes involved in the inflammation of the latter. The antrum seems for a time to retard the outward march of the inflammatory attack, if the attack is not repulsed here, it may go to involve the lateral sinus, the dura mater, and finally produce a brain abscess.

Hence the order of operating, first the middle ear, then the antrum, then the lateral sinus, and lastly the brain.

I may say in passing, that for years it has been my habit to treat the middle ear when diseased,

more heroically and radically than is generally advised.

When you are dealing with a stinking granulating middle ear, moderate curetting or an application of pure carbolic acid can do no harm, but much good. Syringing under these circumstances is generally ineffectual.

The next and last cases I wish to bring forward are two, in each which amputation of the thigh-bone was performed.

Case 1.—Myeloid sarcoma of the lower third of the right femur.

George G. aged 18, admitted into the Victoria Hospital on October 13, 1890.

History. He first complained of pain in his leg in July, three months ago; he continued to go about, was harvesting in August, and hop picking in September, although he suffered pain during both these months.

The leg began to swell in the middle of September; this was then thought to be due to rheumatism.

On October 8th, he was obliged to take to his bed; when in bed his leg was noticed to crack on movement, and the swelling had rapidly increased.

On admission. At the lower third of the right femur, there is a roundish elastic swelling, which on gentle pressure gives out a marked crackling sound. The temperature is 100° F., but no further sign or symptom apart from the leg is noticeable.

On October 15, the thigh was amputated in the upper third by antero-posterior flaps. The patient nearly succumbed from shock after the operation. He left on December 11th, convalescent.

Condition of Diseased Parts. The lower third of the femur was globularly expanded out to the size of a cocoa-nut, the bony walls of the cavity being of paper-like consistence, the cavity was filled with broken-down blood clots and fungoid-looking masses of growth varying much in size.

Case 2.—Fracture of the right thigh, just above the condyles—slightly compound.

Emma R., aged 27, lady's maid. On Friday, May 8th, 1891, fell down some stone steps in the dark, at an hôtel in Boulogne. The leg was immediately put up in a stiff gum and starch bandage from the foot to the upper part of thigh inclusive, by a French surgeon. The next day she was admitted into our local hospital, when we found the lower part of the right thigh and knee-joint much swollen, there was a wound in the skin just above the natural position of the patella in which were two silver-wire sutures. The thigh was not thought to be broken, there being no shortening ! A back splint with a foot piece, extending upwards as far as the middle of the thigh, with two side splints, were applied.

On Thursday, May 14th, the sutures were taken out because of some pus appearing, and the next day two drainage tubes were put in under an anæsthetic.

On May 18th, an anæsthetic was again given, two tubes were put in, on each side of the joint two inches distant from the original wound.

Friday, May 18th, an anæsthetic was given for the third time, and a long tube put right through the back of the joint, the leg being placed in a "Macintyre" splint. Since the patient's admission her temperature had ranged between 100° F., and 102° F., and on two occasions had reached 103° F.

It was decided on June 7th, a month after her admission, to examine again and amputate, if necessary. The latter procedure was carried out, the amputation being done by antero-posterior flaps in the lower third of the thigh.

Condition of Injured Parts. There is a transverse fracture of the lower part of the femur with a vertical one extending down between the condyles, the upper fragment was driven over the lower, and in contact with the under surface of the patella. She left convalescent on August 8th.

Remarks. The first case is remarkable, in that the boy is still alive. Myeloid sarcoma of the bone is an exceedingly malignant disease, and by statistics in more than 80 per cent. of the cases operated on, the disease returns within a year. The condition of the bone was most typical of this form of sarcoma, and the well-known sign of egg-shell crackling was very well marked. It is noticed that the boy nearly succumbed to the shock of the operation.

Operations have almost lost their former terrors. The fears of septicism, of hæmorrhage, and of anæsthetic dangers, have nearly ceased to exist among operators; but shock still remains as a factor in all large operations. This must receive more thought; in fact, is receiving it now. The points that require attention seem to be that the patient should be maintained during the operation in a uniformly warm atmosphere, the body temperature reserved by wrapping the patient in cotton wool, or surrounding him with hot water bottles or pipes. Stimulating rectal injections may be given just before and immediately after the operation; and, again, I believe, strychnine injections, under suitable circumstances, will be found most beneficial, more so than ether or brandy, as these last are so evanescent in their action.

The second amputation case recorded here is a very sad one—the cause of the accident comparatively a slight one, but the result most dire. The skin wound was a small one, but this was sufficient to start a septic condition which we were unable to control. Possibly if the case had been thoroughly irrigated from the commencement of the injury the result might have been different. The condition of the parts injured, and the locality of the mischief, both rendered preventive treatment inefficient.

CLINICAL
DEMONSTRATION OF CASES.

Given at the Monthly Meeting of the North-West London
Clinical Society, held at the North-West London
Hospital on Wednesday, Jan. 9, 1895.

FRED. DURHAM, Esq., F.R.C.S., in the Chair.

Erythematous Ulceration of the Extremities.

MR. JACKSON CLARKE exhibited a case of a little boy
who had been ill for some time with pain about the hip, and
had been treated during the preceding twelve months for hip
disease. There was no deformity, and what now remained
was quite latent. He had been kept at rest at home, and
during the course of treatment cutaneous ulcers had ap-
peared, and had been in the habit of appearing each
winter. On his hands and lower extremities the ulcers were
most frequent. The patient complained of pain in the
knee-joint; he had a feeble circulation, and always cold
extremities in winter. The first question in the diagnosis
was whether this might not be a case of Bazin's disease.
One lesion appeared, and was watched, and revealed first a
minute red ulceration, extremely tender, of which the middle
became vesicular without much tension at first, the central
vesicle being of a blueish colour, surrounded by a whitish
ring, and round that an erythematous zone. In the main
these might be put down in ordinary parlance as neurotic
vaso-motor lesions, which are closely allied to chilblains.
In the rarer form of erythema iris one central vascular
lesion becomes surrounded by secondary vesicles, each one
having rainbow alternations of colours. This was a com-
moner kind. As a rule it does not go to ulceration, but
stops short at pigmented spots without a scar. Mr. Clarke
said his treatment was keeping the extremities warm and
feeding the boy up well. All that could be done in that
way was by a mixture of cod-liver oil and iron. He thought it
was of some interest to bring together the group of different
skin lesions from Raynaud's disease to this form and then to
erythema multiforme. He would say the case had not to do
with herpes zoster, nor with tuberculosis, but had to do
with vaso-motor phenomena.

In reply to the Chairman's suggestion that the lesions were
ulcerated chilblains, Mr. Clarke said, had he not watched
the case he would have thought them of this character,
but from their evolution the case was typical of herpes iris.
Still the ulcers were closely allied to chilblains.

Hereditary Congenital Spastic Paralysis.

DR. GUTHRIE exhibited two children to illustrate this
disease. Three children of the family—the first, second,
and fourth—suffered from the disease, the third and fifth
being perfectly healthy. One of the boys was brought to
Dr. Guthrie because he was "always falling about." The
gait was found to be unsteady, and he had exaggerated
knee-jerks. The two healthy children took after the
mother, those affected taking after the father; therefore, he
regarded the case as hereditary. The father was a packer,
38 years of age, and said he had never had a day's illness
in his life; but the wife stated that for the last ten or twelve
years he had had something very peculiar about his gait.
The man himself said he could walk ten or twelve miles
without fatigue. The father was asked to walk across the
room, and the peculiarity in his gait was noted. Dr.
Guthrie showed the exaggerated knee-jerk and ankle clonus,
but the ankle clonus was not present in the children. He
thought it was extremely difficult to form an opinion as to
the exact site of the underlying lesions, but it appeared
that they did not threaten life or health. The most pro-
nounced case he knew of spastic paraplegia was that of a
blacksmith, who was able to do his work quite well, as well
as other exercises, and he had the ailment for years, during
which his walk always attracted attention. Neither the
children in the cases before them nor the father had any

bladder or rectal troubles whatever. The elder child was
born quite easily and presentation was normal, and it was
not considered there was anything the matter with him
until about 2 years of age, when he showed no signs of
being able to walk. He could not yet walk, but could
shuffle about with assistance. The case was clearly one of
spasm and paralysis. The muscles below the knee were
wasted, and the whole limbs were very cold and blue.
Besides knee-jerk exaggeration, there was a rigidity of the
knee and a tendency to cross-legged progression. There
was, in addition, weakness of the muscles of the back,
which caused him to sit huddled up. There was very little
or no spastic condition of the arms. The face wore a
certain vacant, almost imbecile expression, yet he was not
an idiot, but rather intelligent, and he had a liking for
putting boxes together and hammering in nails. His speech
was somewhat affected. The case of the girl was rather
a contrast, as her leg was paralytic. She also had exag-
gerated knee-jerk and considerable rigidity of the limbs,
but no ankle clonus. She had no ataxy. Dr. Guthrie said
hereditary cases had been published lately of lateral sclerosis,
but the difference between those and these was that in the
published cases the symptoms did not occur until usually
towards the end of the first decade, whereas in these cases
the symptoms were congenital. The absence of ankle clonus
was remarkable, and Dr. Gowers explained such absence
by the assumption that the reflex arc at the lowest part of
the cord was intact.

DR. CAGNEY believed that cases of congenital spastic
paraplegia were more common than was supposed, and
when our minds were open to see them they would be
oftener recognized. It was true that the earlier observations
had shown this condition beginning late in childhood; but
there were cogent reasons for the expectation that the point
of junction anatomically and functionally between the fibres
of the pyramidal tract and the motor cells in the cord
should be a weak spot at an early period of development.
From this point of view Dr. Guthrie's cases were of extreme
interest. They were hardly less so from the absence of ankle
clonus, though such absence was common enough. His own
view as to the absence of ankle clonus in such cases was
that this was further evidence already pretty strongly against
the hypothesis that ankle clonus and the knee-jerk depended
on the same mechanism as ordinary reflexes.

DR. GILL agreed that spastic paraplegia in very early
life was not uncommon. He could recall three cases which
he had had under his care in the St. Pancras Infirmary, and
he believed he had seen others.

Nævi cured (a) Spontaneously, (b) by Electrolysis.

DR. NATHAN exhibited a boy with a patch on the back
of his head, the result of a swelling, which was present at
birth. The birth was perfectly easy, was over very quickly,
and he was born in a normal position. The swelling was
about the size of an egg at birth, and, according to the
mother, was soft, and apparently very much like what is
felt in a cellulo-cutaneous nævus. He was unable to say
whether it was a nævus. The boy now simply showed an
atrophic patch of skin, which was quite devoid of hair until
three years ago. The hair follicles were very few. The boy
came under observation through being sent home from
school, owing to ringworm; but there had been no ring-
worm, and no treatment of the head.

The other case was one of ordinary nævus, affecting the
pinna of the right ear, and extending backwards, causing
swelling, pushing the ear forward, and occupying all the space
between the pinna and the scalp behind. He went to St.
Mary's Hospital four months ago, and had been treated by
electrolysis three times, the last having been completed a
week ago. The result had been fairly good. Cases of nævus
of the pinna lent themselves especially to electrolytic treat-
ment because it would be next to impossible to remove
them by other surgical means without running the risk of
disfiguring the patient.

MR. JACKSON CLARKE said that, as far as he knew, the

first case was unique, and it was a particularly interesting one. He had not encountered mention of such a case in any works on dermatology. The only thing which suggested itself was injury in parturition, but that was merely surmise.

THE CHAIRMAN said he had seen and had treated a great many nævi by electrolysis, and had had some very good results indeed. The only thing which suggested they were all agreed that when a nævus was so isolated that it could be removed by the knife, the knife was to be preferred to the needle or the hot point. He thought Dr. Nathan was much to be congratulated.

Malformation of the Chest.

DR. CAMPBELL, in showing a man with emphysematous chest, remarked that he did not bring it as an instance of a rare disease, but as a rare instance of a common disease. He took it the function of the Society was not merely to show remarkable instances of uncommon diseases, but interesting instances of common ones also. The case was a very good type of barrel chest; the ribs and the shoulders were high and the neck thick, the diaphragm low, the heart low and deep. The patient was now fairly comfortable; he showed no marked signs of dilatation of the right heart or dilated veins. One cause of the secondary affection was interference with the pump-like action of the lungs during inspiration and expiration; the blood was sucked from the large veins into the right heart, and from the right heart through the veins into the left heart, and it therefore followed that if there was any interference with the respiratory movement the blood got dammed back on the large veins. In the treatment of such cases, Dr. Campbell believed some good had resulted from compressed air treatment. Dr. Theodore Williams, in his book on aërotherapeutics, alludes to the advantage which this treatment brought about in such cases. The general cause was chronic bronchitis.

Multiple Osteotomy for Deformity.

MR. JACKSON CLARKE showed a case of a little girl on whom osteotomy of the femur and osteotomy of the tibia had been performed on McEwen's method, and, though the result was not highly artistic, it was one of functional adequacy. A photograph of the case before the operation was exhibited, showing the case to be one of greatly exaggerated ricketty curvature. The bones had held up very well. Before the operation the patient could hardly walk. The muscles were not yet well developed, but she could now begin to toddle, and when she got the strength of the muscles back she would do pretty well. Mr. Durham kindly tried her in bed with splints, but the bones would not yield to splints. McEwen had made osteotomy a very beautiful operation, and it had yielded good results. He hoped to show the case again six months later. He believed the parents would be able to carry out his directions, and he looked for the muscles to keep the bones in place. He believed osteotomy enabled them to prevent a recurrence of the deformity, or else he would not dream of having an operation.

MR. GORDON BRODIE spoke in commendatory terms of the work of Mr. Clarke in the case.

Exophthalmos.

MR GORDON BRODIE exhibited a boy, 7½ years old, with unusually prominent eyes. There was no enlargement of the thyroid, and no rapidity of the pulse. There was malformation of the bones of the head. The boy also had a very peculiarly shaped chest. The sternum was remarkably enlarged, and it could be traced backwards almost at right angles so as to press on the trachea. It was a question whether the condition might be due to some maldevelopment in which the venous system had been pressed upon, producing venous congestion of the parts in uterine life. He had removed some adenoids, but the child still had difficulty in breathing.

DR. CAMPBELL commented on the fact that the child had nasal obstruction and high palate, and this latter went with adenoids. He had no doubt this nasal obstruction interfered with the development of the entire cranium. On the rarifaction of the air in the nasal cavities the hard palate got pushed up, and that interfered with the entire development of the cranium. He looked on it as a good object lesson on the interdependence of parts in the human body.

MR. CLARKE thought that to believe the shape of the head was caused by the adenoids required a good deal of imagination. He thought it was rather due to a mechanical uterine condition.

DR. COALL commented upon the adequacy of nasal obstruction to produce some of the appearances which had been attributed to it in this and other cases, and expressed the opinion that too much weight had been attached to that condition.

DR. CAGNEY regarded the changes both in the head and the chest as the result of rickets. The facial expression, connected as it was with adenoids, gave further confirmation of this. Such a case well illustrated a chapter in the progress of knowledge. Ricketty children were long known to suffer from laryngismus stridulus, and this had been referred to an irritable state of the nerve centres caused by venosity of the blood, the latter being ascribed to the yielding character of the chest walls. Laryngismus was now attributed with more probability to direct suffocation by adenoids, and it was undoubtedly relieved by removal of the latter. An interesting problem remained, namely, as to the connection between the morbid proliferation of lymphoid tissue and the nutritive defect of bones and other structures.

A Case of Myelitis.

DR. CAMPBELL showed a case of recovery from myelitis which he believed was somewhat rare. The patient was sent to the hospital absolutely paralysed and almost incapable of moving his legs. The touch was perfectly normal, but sensibility to temperature and pain were distinctly affected. Pain sensations were diminished, and cold was always felt as heat. He had incontinence of urine, and the facts pointed to involvement above the lumbar enlargement, and he is now practically cured. The usual causes of this affection were exposure to cold and strain. He was put to bed, and given a large dose of iodide of potassium. They strongly counter-irritated the spine by applying a number of blisters; this he considered the right course in all acute cases of spinal disease. He remembered a case when the counter-irritant was too strong, and left a scar all up the spine; but the patient got better, and that had much impressed him. A man should be sent about the world with a scar rather than as a cripple.

DR. COALL related an instance of cure in acute myelitis by the prompt use of blisters and ergot.

Hæmatoma of the Pinna.

MR. JACKSON CLARKE showed a man of about 30 years of age with a hæmatoma which fluctuated. It looked like an effusion of blood between the perichondrium and the skin of the ear. Suspicions had been thrown on psychical alienation, but no history of that had been traceable in this case. It appeared to be quite spontaneous. It was at first only half the present size, and pain was only present in it occasionally. If there was much pain he supposed the treatment would be to apply ligatures, and if more pain still, to excise the tumour. As this tumour had extended he thought he would let out the contents, and then apply some pressure to prevent its collecting again.

MR. RIDLEY asked whether a tendency to hæmorrhage had been found in other parts of the patient's body.

MR. BRODIE mentioned a case somewhat similar which yielded a serous fluid on being punctured.

MR. CLARKE said he always made inquiries as to a tendency to hæmorrhage and a history of hæmophilia before he operated in any case, and he felt sure the course he had mentioned would be the best.

DR. ATKINSON (for MR. MAYO COLLIER) showed a case of hallux rigidus, and also one of polypus of the frontal sinus.

A CLINICAL LECTURE

ON

RESEMBLANCES & DIFFERENCES IN DISSEMINATED SCLEROSIS, LOCOMOTOR ATAXY, AND FRIEDREICH'S ATAXY.

Delivered at the National Hospital for the Paralysed and Epileptic, Dec. 19, 1894, by

JAMES TAYLOR, M.A. Edin., M.D., M.R.C.P.

GENTLEMEN,—I propose to-day shortly to de-scribe or demonstrate three cases of somewhat allied diseases, to point out their symptoms, to tell you the history so far as it has gone, to contrast and compare those cases, to say a few words about their morbid anatomy, more especially as regards the spinal cord, and finally, to allude to the most suitable treatment. The three diseases of which I propose to speak, and of which I have examples here to-day, are disseminated sclerosis, locomotor ataxy, and Friedreich's or hereditary ataxy.

Case 1.—The patient before you is a man 33 years of age. He first came to see me two years ago, and the history then was that a year pre-viously he had had some difficulty in using his right hand, and that he had not been able to follow his usual occupation as a farrier on account of that difficulty, which consisted especially of unsteadi-ness of the right hand. This difficulty had gone on increasing during the year previous to that in which I saw him, and had gradually affected the left hand as well. Along with this there was developed a certain amount of difficulty in walk-ing, so that he occasionally struck his foot on the ground and was unsteady, but not sufficiently so to cause him at any time to fall down.

Then, for four months before he came under observation he had had considerable difficulty in articulating; he had been unable to speak with that clearness and distinctness with which he had been used to speak, and this difficulty was very much aggravated during any excitement. At the same time there had also developed a certain amount of emotional instability. When he came under observation his condition was very much what it is now; he had a certain amount of un-steadiness of the hands, which was considerably aggravated in carrying out any action—as, for example, trying to touch his nose with his finger. The same difficulty was present in the left hand, and in a more aggravated form, although when he was first seen it was more marked in the right hand than in the left.

With regard to his walking, you can see that he walks in a curious manner, and that he oc-casionally strikes the ground with his foot. Although there is a certain amount of spasticity present, that is not so clearly marked as it usually is, and it appears that the difficulty in walking depends really more upon some tremor which is present in the trunk and legs. You will notice that the upper part of his body shakes considerably, and the tremor of the head is very well marked indeed. Before he came under observation he complained of a certain amount of difficulty with his vision, and when I examined his eyes I found there was extremely well-marked nystagmus, which was very much aggravated on deviation to one or the other side. Besides this he had extremely pale discs, and the visual acuteness was reduced—*i.e.*, there was a slight degree of optic atrophy. Then as regards the condition of the reflexes, he had exag-gerated knee-jerks on both sides. He never had any ankle clonus, but he seems to be developing perhaps a slight tendency to it now. You will see on looking closely well-marked nystagmus in both eyes.

· I think, as a rule, you will find that the pecu-liarity in articulation described as occurring in dis-seminated sclerosis is the syllabic utterance—that is to say, each syllable of a word is separated from the following syllable. In my experience there is another difficulty of articulation which is still more frequent, and that is the difficulty in clearly pro-nouncing the terminal syllables in any word—the patient's articulative muscles seem to become tired before the end of the word is reached, and the final syllable or syllables are pronounced in a much less clear and distinct manner than are the

initial syllables—and that is the kind of difficulty which this patient had and still has.

(The patient here answered one or two questions put to him by the lecturer.)

You see the peculiar articulation very closely resembles what you find in general paralysis. There is one other point with regard to this disease, and that is the mental condition of the patient. It is said that in disseminated sclerosis there is no marked mental change, but in some cases there is a certain amount of it, so that patients who are comparatively helpless take a much more rosy view of their condition and of their circumstances than the circumstances themselves justify. That was not particularly marked in this patient, although he always was in very good spirits, but in certain patients it is a very marked feature of the case—a condition which very closely approaches that which is found in general paralysis. Yet I have never known any case of disseminated sclerosis which did become a case of general paralysis, and that, of course, has to be borne in mind because, although the mental condition approaches that found in general paralysis, still it by no means follows that one condition will pass into the other.

As regards the sensory condition, this patient demonstrated what is the usual condition, viz., that there was absolutely no disturbance of sensibility, no shooting pain, no pain in the back, no pain of any kind; and on careful testing, no deficiency at all in sensibility was detected in any part of the body. Nor had he what is very often present in disseminated sclerosis, a certain amount of difficulty in connection with the sphincters. Very often you find difficulty, or some trouble at all events, in connection with the passing of water; but this patient had no trouble of that kind. Nor in disseminated sclerosis is there any affection of the pupils. There is no loss of the reaction of light, such as you get in certain other conditions to which I shall refer presently.

So that the prominent points in this case, as in all cases of disseminated sclerosis, were tremor, the difficulty of walking, nystagmus, optic atrophy, tremor of the head, the difficulty of articulation, and the absence of anything like sensory change, and together with these symptoms was the exaggeration of the knee-jerk. I ought also to mention that in this patient, as you usually find in disseminated sclerosis, there is no reason whatever to suspect any syphilitic affection; there

was no history of any possibility of syphilis. The patient had three healthy children, and his wife had had no miscarriages; and there was absolutely nothing to which this illness could be ascribed. Very often you find a history of a shock or accident preceding the onset of symptoms; but in this there was nothing of the kind, except that he might have had an occasional knock in the course of his occupation.

Case 2.—This patient first came under my observation six years ago. At that time he was under Dr. Ferrier's care in the out-patient department, but he afterwards became an in-patient. At the time he came under observation, he had suffered from pains and numbness in his limbs for a year. There had also been a certain amount of difficulty in passing his water, and he had had a tight feeling round his waist and considerable difficulty in walking. In his history there was nothing of very great significance; there was no syphilitic history, and nothing which could be twisted into such a history. When he came into the hospital he had, in a slightly less aggravated form than he has now, the difficulty in walking which is characteristic of locomotor ataxy. He walked with his feet widely separated, and with his eyes usually fixed on the ground, in order that he might get all the assistance available from recognition by his eyes of his actual position. In locomotor ataxy one of the first symptoms is impairment of the muscular sense or the sense of position. He walked in this straddling fashion; he had the numbness of the legs, and a certain amount of impaired sensibility, delay in the conduction of thermal and painful sensations, as is often found in the early stage of locomotor ataxy.

On carefully testing the feet now you will find that light touches are perceived with a fair amount of acuteness; but on testing with some sharp point and with heat or cold, more especially with heat, you will find there is a very marked delay in the conduction of the painful stimulus, and also of the thermal stimulus. So that when you put a hot point on the patient's foot, he first of all feels the touch and then, after an appreciable interval, says it is a hot touch. It is very easily demonstrated in the great majority of cases. It is not always recognized that you may have delay in the conduction of thermal and painful stimuli while the sensibility to ordinary touch is quite well preserved. While he had this ataxic walk and numbness, and the delay in conduction of thermal and painful

stimuli, his pupils were in that condition which is not usual in locomotor ataxy, that is, they reacted readily both to light and during accommodation; *i.e.*, he had no Argyll-Robertson phenomenon. But he had shooting pains, so-called lightning pains, which are so characteristic of *tabes*, pains in different parts of the legs coming on suddenly. They were very severe, but only came on certain days, and in the interval he was perfectly free from them.

Then he also had, as I have said, a certain amount of difficulty with the sphincters; he had some difficulty in passing water, and a slight degree of difficulty in controlling the sphincter of the bladder. He also had a similar trouble in connection with the bowels, more especially after taking medicine. As regards the condition of the knee-jerk, there are certain cases of locomotor ataxy which apparently begin high up in the spinal cord, not associated with any difficulty in walking, and in which the knee-jerk for a certain time is still retained, but those are exceptional cases. In the majority of cases the knee-jerk, as it is in this patient, is completely lost. I might also mention that this patient has no nystagmus and no optic atrophy. You do find optic atrophy in tabes, more particularly in those cases to which I have just referred, in which the disease seems to commence high up in the cord; but this patient has shown no sign of anything of the kind, and now, of course, it is not likely to be developed. As regards his walking, you will see he walks in a very characteristic ataxic fashion. He is only able to walk in that way.

Case 3.—The next case which I show you is an example of a very rare form of disease, the so-called hereditary ataxy, usually known as Friedreich's ataxy. This patient came under my observation first of all about four years ago, when he was a patient of Dr. Hughlings Jackson, and complained of very great difficulty in walking. This difficulty had been coming on for several years before this, and had been slowly becoming worse. He also complained of a certain amount of unsteadiness in his hands and head, and some deformity in his feet, which he thought interfered with his walking, and he also complained that one of his shoulders had been growing out.

He had also noticed that his articulation had been peculiar; he had not been able to speak so clearly or so distinctly in the last few years as previously. He is now 20 years of age. When I saw him first he was about 15 or 16; the trouble had commenced some years before that.

At that time he had the difficulty in articulation which I have described, somewhat resembling the articulation in disseminated sclerosis. He also had a difficulty in walking. He had no pain, no trouble with his sphincters, no sensory impairment at all, but he had lost both knee-jerks. He had also a very marked deformity of his back. You will see the extremely marked lateral curvature which he has. It is rather more aggravated than it was when I saw him first. Besides that he had a "hump foot," a deformity characteristic of Friedreich's disease. The deformity was very marked. Those two deformities, or at least one of them, are found in the great majority of cases of Friedreich's or hereditary ataxy.

(The patient here answered a few questions put to him by the lecturer.)

Unfortunately the words which I got him to say have not brought out the peculiarity of articulation which you find in these cases—it is not very marked, however, in this particular case. Then one condition of his eyes is noticeable—he has a slight degree of nystagmus, as we often find, but not so marked as that usually present in disseminated sclerosis. In this case it is very slight, and if you examined his eyes you would probably say that it was not present. It becomes more marked, of course, when his eyes are tired.

I should now like to say a few words about the differential diagnosis of these cases we have seen —disseminated sclerosis, locomotor ataxy, and Friedreich's ataxy; and, first, as to disseminated sclerosis and locomotor ataxy.

The conditions are to be distinguished, first of all, by the gait. As you saw, in disseminated sclerosis, the gait is of an entirely different character from that which is found in locomotor ataxy. In the latter, ataxic is the best word to describe it; in the former, the peculiar difficulty in walking is dependent in a certain degree upon tremor in the muscles which are concerned in walking, and also upon the spasticity or stiffness. The marked feature in the gait in the case of disseminated sclerosis, as we saw, was the unsteadiness; the marked feature of the walk in locomotor ataxy was the ataxic inco-ordination—unsteadiness in one sense, but in quite a different sense from that of the disseminated sclerosis.

Then again, as regards articulation, the patient in locomotor ataxy had no difficulty in articulation,

and the articulation was not disturbed; that is to say, he spoke like an ordinary man. But the patient with disseminated sclerosis, as I think you must all have recognized, spoke in a very peculiar manner. He had a difficulty apparently in speaking distinctly and clearly, and, as I have said, the articulation resembled very closely the blurred and defective articulation which you meet with in general paralysis.

Then as regards pain and the sensory condition generally, disseminated sclerosis and locomotor ataxy are to be distinguished, because in locomotor ataxy pains are a very common feature in the great majority of cases, and sensory impairment is nearly always present. In disseminated sclerosis you have neither pains nor sensory disturbance of any kind. The pupils in locomotor ataxy usually show impairment or abolition of the reaction to light; for, although the case we have here of locomotor ataxy did not show this so-called Argyll-Robertson pupil, that is an exception, and this condition of pupil is never present in disseminated sclerosis. This gives a fairly constant mark of distinction between the two diseases.

Then again, as regards mental condition, it is true that in locomotor ataxy there may be, as it were, grafted on the disease, the condition known as general paralysis of the insane. Cases of *tabes* do develop into general paralysis, or perhaps it would be more correct to say that in cases of general paralysis, posterior sclerosis may be an early symptom. In disseminated sclerosis I have never seen any condition of general paralysis of the insane develop, but in a certain number of cases you find a mental condition which closely approaches that which you find in general paralysis, although not quite so marked. Then, as regards the reflexes, these constitute a very distinct mark of difference between disseminated sclerosis and locomotor ataxy, and the reflex to which we attach most importance is the knee-jerk.

As regards knee-jerk, it is practically always absent in locomotor ataxy; it is nearly always exaggerated in disseminated sclerosis, never being absent, and often ankle clonus is associated with it. As regards the organic sphincters the condition is usually the same in the two diseases; in disseminated sclerosis you usually have early in the disease a certain amount of difficulty either in retaining or in passing water. In locomotor ataxy you have practically the same condition in the early stage, a certain amount of difficulty in

holding or in passing it. Then, as regards the condition of the optic discs, you often have practically the same condition, viz., a certain amount of optic atrophy. But in my experience optic atrophy is much commoner in disseminated sclerosis than in locomotor ataxy. Still, the presence of optic atrophy scarcely helps to inform you to which category the particular case belongs. In distinguishing between the two, you have to depend upon the gait, upon the character of the articulation, upon the presence or absence of pain, upon the condition of the pupils, and most of all upon the condition of the knee-jerk.

Then, as regards the distinction between disseminated sclerosis and Friedreich's ataxy. In the latter is the age as a guide; it is very rare, possibly unknown, for the symptoms to commence after the age of 20—they nearly always commence before. Then again, you have the history of the patient. Friedreich's ataxy is essentially a family disease, and in the majority of cases you will find that there is a family history of some similar condition of paralysis.

This patient, I ought perhaps to have told you, has a brother two years older than himself, who has been a patient under Dr. Jackson's care here, being practically a counterpart of the patient here to-day. He is also suffering from Friedreich's ataxy. You generally find that some relation has suffered from a disease which is probably the same. On the other hand, of course there may be—I have come across more than one case of this kind—no family history to guide you to the diagnosis of Friedreich's ataxy, yet in which the condition is sufficiently distinct to make the diagnosis by no means doubtful.

Then there is in the condition of the sphincters a useful distinguishing point between disseminated sclerosis and Friedreich's ataxy. The sphincters in disseminated sclerosis are nearly always affected; in Friedreich's ataxy I have never seen them affected, and, as a matter of fact, affection of the sphincter will not be found except in the very last stages, when there is no difficulty at all with regard to the diagnosis. Then, as regards the reflexes, you have the same point of distinction as between disseminated sclerosis and locomotor ataxy. In Friedreich's ataxy the knee-jerks are nearly always absent. Then, also, in Friedreich's ataxy there are the deformities,—the deformity of the back and the deformity of the foot,—usually one or the other

of them is present; in disseminated sclerosis there is never anything of the kind, at all events, if there is, it is not an essential part of the disease, whereas in Friedreich's ataxy it occurs with such regularity that it must be regarded, to a certain extent, as part of the disease.

Then again, in disseminated sclerosis there is very often a slight degree of optic atrophy, not sufficient really to interfere very much with vision. Now, in Friedreich's ataxy there is never optic atrophy. If it is present it is a very anomalous case, and it is certainly not Friedreich's, but a disease different from, although perhaps allied to, the ordinary Friedreich's ataxy. A certain family disease has been described which is associated with optic atrophy, but from the symptoms it is scarcely likely that the disease is the same, at all events. In all the cases of Friedreich's ataxy which I have seen—and I have seen about twenty—there has never been the slightest degree of optic atrophy present.

Now as regards the resemblance between disseminated sclerosis and Friedreich's ataxy. You noticed that the patient with disseminated sclerosis had a certain amount of tremor of the head. There is very often a similar kind of tremor present in Friedreich's ataxy, only in Friedreich's ataxy the tremor more closely resembles what is found in chorea, so that I have known a case of Friedreich's ataxy mistaken, in the out-patient room, for a case of chorea.

Then as regards the gait. This patient with Friedreich's ataxy is scarcely able to walk, but if you did see him walk you would see that he had a very great difficulty in keeping one line; he will stagger about and reel as he walks. Whereas the patient with disseminated sclerosis simply walked as you saw, with that tremulous gait which is to be expected when there is some tremor in the muscles which are concerned with locomotion and with the spasticity which is usually present. The gait of Friedreich's ataxy is one more of in-co-ordination, and it is described as resembling the reeling gait which there is with cerebellar disease.

As regards articulation, there is a certain amount of resemblance between disseminated sclerosis and Friedreich's ataxy. The articulation in each case is blurred and indistinct, and it is of that curious character to which I have referred, so that it does not always constitute a very distinct mark between them, but is rather one of the characteristics in which the two diseases may be said to resemble each other.

Then as regards nystagmus. You nearly always find nystagmus in disseminated sclerosis and in Friedreich's ataxy, but in Friedreich's ataxy the nystagmus is not nearly so marked, and is not of that oscillatory character which it is in the other case. There is no impairment in the reaction of the pupil in either case, and in both you have the absence of symptoms which constitute the distinguishing marks between these diseases and locomotor ataxy.

Now just a few words as regards the distinctions and the resemblances between Friedreich's ataxy and ordinary locomotor ataxy. Of course, it may be difficult if a case comes under your observation of a man of 33 years of age, to know whether it is a case of ordinary locomotor ataxy or a case of Friedreich's ataxy; but the history will always tell you, because in Friedreich's ataxy you will have a history at all events that the disease commenced before the age of 20, whereas in locomotor ataxy you practically never have the disease commencing before 23. The character of the family history, to which I have already alluded, will also assist you. Then, also, pains and sensory affections are present in locomotor ataxy, and are absent in Friedreich's ataxy, whilst deformities are present in the latter and absent in the former. Then as regards the pupils. In locomotor ataxy there is usually the Argyll-Robertson phenomenon present, although it *may* be absent, as it is in this patient. And as regards the sphincters. They will never be interfered with, at all events not in the early stages, in Friedreich's ataxy, whereas in locomotor ataxy it is the rule for a certain amount of difficulty to occur in the early history of the disease. As regards the reflexes, more especially the knee-jerk, of course the cases are practically similar; both in locomotor ataxy and Friedreich's ataxy it is the rule to have the knee-jerks abolished.

Now as regards the pathology. In considering pathology, I shall simply glance at the condition of the spinal cord. I shall not go into the ultimate pathology of the diseases, but only into the pathological anatomy, especially as it affects the spinal cord. In disseminated sclerosis there is, of course, a scattered sclerosis throughout the spinal cord and throughout the brain. I show you here a specimen which Dr. Colman has prepared, which will show you much more dis-

tinctly even by the naked eye what is meant by scattered sclerosis than my description can do. In locomotor ataxy, the essential thing is the sclerosis in the posterior columns. Here I have a section of the spinal cord which shows this very well indeed. It is a section from the cord of an old patient who died of locomotor ataxy—he suffered from the disease for twenty years—and it shows very markedly the sclerosis in the posterior columns of the spinal cord. This will show you quite distinctly what the condition was. As regards Friedreich's ataxy, there is in that disease sclerosis of the posterior columns as the most marked thing, and there is also the scattered sclerosis round the different areas of the spinal cord, and not only of the posterior. That is to say, the sclerosis in the posterior columns is very well marked indeed, and there is also, in the lateral columns, and even in the anterior columns, more especially at the margin, a certain amount of sclerosis, not nearly so deep as in the posterior columns. So that in Friedreich's ataxy there is really the kind of sclerosis which there is in this specimen from another disease. This cord shows very well the kind of sclerosis there is in Friedreich's ataxy, and, apart from the clinical history of the case, it might well be diagnosed as a case of Friedreich's ataxy. I have those three specimens here to show you the pathological condition which is found in the spinal cord in these cases.

And now a few words with regard to treatment. In disseminated sclerosis you can only mitigate the disease, you cannot cure it—you may prevent it from advancing by care and rest, but it cannot be cured. Then as regards rest. I believe it is a very good thing when you recognize a case of disseminated sclerosis, to put the patient in bed and keep him there for two or three months absolutely at rest. The cases in which I have known the best results to be obtained have been treated in that way. Another thing that often does good is the combination with the rest of massage, and you may give Faradism also. You will then expect a certain amount of benefit, at all events in the general condition of the patient, and you must recognize that the progress of the disease will depend upon the general condition, both physical and mental, of the patient. Nothing is more certain than that disseminated sclerosis is very much aggravated indeed by mental worry, and in some cases it seems to be actually produced by this cause. At all events, a great many cases

of disseminated sclerosis have the history that just before its commencement there had been some violent mental emotion or mental disturbance, and it is essential in carrying out the treatment of disease to recognize this fact, and to prevent anything of the kind from occurring so far as it is possible. Keep the patient under the best condition of physical and mental rest that is attainable.

As regards drugs, I cannot say I know of any which have any practical effect upon disseminated sclerosis. Most tonic drugs will, by improving the general condition, tend to improve the special condition, but there is no special drug that I know of which has any peculiar effect upon the disease. Hyoscin I have seen tried, and it seems to modify the tremor to some extent; but the improvement with regard to tremor has been associated with general depression, so that the patient's mental condition was certainly worse, and a condition was produced which was not any better than the condition was before the tremor had been modified, and as soon as the hyoscin was stopped the tremor again became troublesome. So much for the treatment of disseminated sclerosis.

Now as regards *tabes*. There is no doubt that rest is a very great factor, and rest in bed I believe to be efficacious in cases of this disease. I do not think the patient ought to be kept solely at rest in bed; I think there ought to be combined with that very active treatment of the spine by counter-irritation by means of the actual cautery. The best results I have seen in tabes in hospital have been in cases in which the cautery has been used very freely indeed, the treatment being accompanied by rest in bed. There are certain drugs also which are said to be useful—*e.g.*, nitrate of silver. I cannot say I have seen it do any good. I have seen patients improve while taking chloride of gold and soda, but whether that was a coincidence or whether it has had any particular effect, of course it is impossible to say. I may say I have seen one case improve under that treatment very markedly. Another drug has been lately introduced by Dr. Gowers—viz., chloride of aluminium, and I have seen that do good undoubtedly. In three cases I have seen distinct improvement, and in a good many more the patients have certainly not become worse when taking that three times a day. They have no difficulty in taking it; it is quite soluble, and I am inclined to attach rather more importance to that

drug in the treatment of *tabes* than to any other. Of course it may be that in those cases which I have seen improve it was merely a coincidence, but even coincidences are encouraging in a disease which is so difficult from the point of view of therapeutics as locomotor ataxy. The two factors in the treatment of locomotor ataxy · to which I should attach most importance are the use of rest with counter-irritation of the spine and the administration of chloride of aluminium. Other conditions, of course, such as retention of urine, must be attended to, and the catheter used if necessary. As regards Friedreich's ataxy, I am afraid we cannot expect to benefit this much by drugs. Of course the patient's health must be carefully looked after, and he must be put under the best conditions possible, but beyond that there is nothing to be done. It is a very hopeless disease; it always becomes worse.

You are probably aware that mechanical treatment by means of suspension has been tried in locomotor ataxy especially, but also in the other diseases to which I have alluded. Our experience of this treatment here has been very extensive, but it has not been such as to allow us to attach any importance to it.

A CLINICAL LECTURE

ON THE VALUE OF

EXCITING ASEPTIC CELLULITIS

IN THE TREATMENT OF

Obstinate Inflammatory Affections of the Urinary Organs.*

By E. HURRY FENWICK, F.R.C.S.,

Surgeon to the London Hospital and to St. Peter's Hospital for Urinary Diseases.

GENTLEMEN,—My title and the deductions I have drawn from clinical and pathological observations are open to criticism, but the gist of the matter, I am convinced, is worthy of some consideration. The surgeon of to-day strives at the rapid and aseptic healing of all wounds, and is justly blamed if an incision which he has made through the unbroken skin does not promptly unite without suppuration. I believe, however, that there are many chronic or obstinate inflammatory conditions of the solid viscera, which may be materially benefited by cutting on to them through the parietes of the body, exciting suppuration of a benign type in the cellular tissue of their immediate neighbourhood, and by draining off through the vessels of this layer the products of inflammation. I have been accustomed to term this method of dealing with chronic visceral inflammation, "*aseptic counter-irritation,*" for it is of course nothing more or less than a more accurate and more energetic method of applying the seton, blister, or iodine paint, of old-fashioned medicine. I now submit that the surgery of the present day can advantageously substitute for such surface applications the more powerful and effective counter-irritation of an inflamed and drained cellular tissue.

The examples which I bring forward in support of this proposal are taken from urinary practice, but I have encountered cases equally satisfactory in general surgery, notably in the surgery of the liver.

My attention was first drawn to the matter in 1887, when I was suddenly called upon to treat a patient who was suffering from a supposed rupture of the bladder. He had much dulness above the pubes, though the bladder was empty, and a great swelling was detected in the recto-vesical pouch. On laying open the cavity of Retzius by a suprapubic incision, I found the trouble was merely an acute interstitial or parenchymatous cystitis which subsequently was proved to be due to Bilharzial disease.* In a few days the enormous swelling which had previously existed in the neighbourhood of the bladder disappeared, and it struck me that this unlooked-for result was probably due to drainage of the inflammatory products through the suprapubic incision. Since then I have gradually collected some pathological as well as clinical material which convinces me that inflamed cellular tissue exercises a considerable detergent effect upon any solid viscera with which it is in contact, and that these powers have either been overlooked or not thoroughly appreciated by the profession. The pathological effects of suppuration or extravasation of blood into the cellular tissue in the neighbourhood of solid viscera upon the health of the organ is perhaps well exemplified in the kidney. Here the effects of large collections of pus or of blood

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* Subsequently brought before the Medical Society, London, Dec. 10, 1894.

* "Trans. Medical Society," vol. xi., p. 344, 1888.

upon the plumpness of the kidney can be demonstrated either on operation or post-mortem.

It is often a matter of surprise to find how marked are the effects of perinephritis upon the size and consistency of the kidney. In perinephritis not due to disease originating in the kidney, the kidney is often found small and flattened on the anterior wall of the huge sac. It may be contended that this change results from some irritant in the kidney which has affected it besides inflaming the surrounding tissue, and that the atrophy of the organ is the remote result of the original nephritic disease, and not due to the effects of the perinephritis. The following illustration is therefore introduced as being as unequivocal as I have met with:

Enormous subperitoneal hæmorrhage around the kidney, atrophying that organ.

C. F., aged 30, had been treated by Dr. Ogilvie Will for a ruptured urethra, the result of a fall on the perineum from a height of forty feet. Probably the loin was also bruised, for his buttock and side were black for long afterwards.

Apparently, a large extraperitoneal extravasation of blood which surrounded the left kidney, and had raised the peritoneum lining the left iliac fossa, had formed at the time of the accident. This collection however, was partially absorbed, and when the patient came under my care a year after the accident, for the treatment of traumatic stricture, I was unaware that any blood-clot remained. I operated on the stricture by external urethrotomy. Three weeks after, when I thought the patient was nearly well, the temperature began to rise, and then to oscillate, and he complained of pain in the left loin, similar, as he said, to what he had previously experienced shortly after the accident.

I found the colon and sigmoid pushed forward by a large subperitoneal collection of fluid, the appearance of the loin resembling what is noticed in perinephritis. On incising the fluctuant swelling at the anterior iliac spine, I evacuated fully a pint of puce-coloured pus, and massive, decolourised and black blood-clot. Probably the old unabsorbed extravasation had broken down from some septic infection in connection with the external urethrotomy. The enormous cavity which was discovered reaching as high as the level of the splenic position healed rapidly and thoroughly, and the patient was dismissed from the hospital cured.

He subsequently came under my care with

suppression of urine from calculous blockage of the right kidney, and died. He had been in the habit of passing stones from both kidneys, but had had no attack from the left side since the fall referred to. On post-mortem there was no trace of the large left perirenal sac which had contained so much extravasated blood and pus, its situation was merely marked by extreme thickness and discoloration of the peritoneum in this position. The left kidney was very small, perfectly formed, and markedly fatty. Its pelvis contained a few millet seed calculi. Its ureter was patent, and there was no stone impacted in the canal. The renal vessels were small but apparently healthy. In the absence of any other cause, I considered that the extravasation and efforts of absorption had exerted so powerful an effect upon the kidney that it became crippled and atrophic. In cases of psoas abscess I have found the kidney smaller than normal, but I have not been able to satisfy myself that this might not have been due to wasting noticed in tubercular disease. This case seems to me to point to the fact that solid viscera are greatly influenced by suppurations in their neighbourhood.* The effort of the surgeon should be to induce a mild cellulitis, and thus to imitate *in petto* the detergent results which such widespread and violent inflammations induce.

Clinical Observations. I was very much struck with the startling result which a nephrotomy produced on a profuse and chronic unilateral pyelitis. I removed a large median prostatic lobe which had induced left-sided pyelitis in a man about 50 years of age. The catheter, upon which the patient had been absolutely dependent, was dispensed with, but the pyelitis was quite unaffected, and even remained as profuse as ever, though the bladder was drained suprapubically and perineally for a month. A year after the operation the patient returned to me with abscess in the left kidney. This I incised, and the pus in the urine which had often amounted to ½ during the previous two years completely disappeared, and the urine remained clear for four days. This sudden cessation could not be altogether due to absolute paralysis of the kidney from shock, for even if no urine had been secreted by the diseased kidney, and the ureter had remained unflushed, the pus would still have made its way in some quantity

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* Much sound evidence on this point could be collected by examination of the solid organs of the pelvis after hæmatocele, or of the kidney in psoas abscess.

into the bladder by gravity, and have appeared in the urine. Probably the drainage of the kidney and its surroundings checked the secretion of the pus abruptly, in the same way that an acute epididymitis causes a gleet to temporarily disappear. The pus gradually reappeared, but never in the same quantity as before. The sinus left by the operation remained open, and a year after the patient died. On post-mortem the pathologists could not find any trace of kidney structure.

I was called upon to deal with a slight gonorrhœal pyelitis, accompanied by much pain in the left kidney. After a year's patient trial of drugs I made an opening in the loin, stirred up the cellular surroundings of the kidney, and drained for a week. The urine cleared permanently, and the pain left him.

I suspect that some cases recorded as cured after an ineffectual exploration of the kidney for supposed stone, are due to the beneficial effects of drainage of the surrounding disturbed tissues.

Examples of the effects of Draining the Cellular Tissue surrounding the Bladder. I attempted some years ago to drain a contracted tubercular bladder of a young man in order to relieve him of the pain and distressing frequency of micturition from which he was suffering. But I never reached that viscus. I found dense adhesions in the pre-vesical tissues; and when I opened what I considered to be the little bladder, I found I had entered the peritoneal cavity. The edges of the peritoneal incision were carefully sutured and the original intention of opening the bladder was deferred for a week. He had so greatly improved, however, by that time, the urine had cleared, pain had diminished, and the irritability had been so much relieved, that I decided to do nothing further. I have seen the patient last week, the improvement has been maintained, but the disease has spread to the prostate and testicle. I should not now advocate opening an advanced tuberculous bladder suprapubically.

A lady was sent to me by Dr. Valentine Rees, of Brecon, with a pyæmic pelvic abscess opening presumably into the bladder; the position of the abscess was doubtful. On cystoscopy the orifice of the abscess was easily detected, and Mr. Woodhouse Braine, my assistant, and I watched for some time the following curious phenomenon of pus being forced into the bladder through a minute opening. On the right lateral wall of the bladder low down, enormously swollen

folds of mucous membrane were seen. In a furrow between two of these succulent rugæ, I suddenly noticed a long, white tapeworm-like body being forced out. It was square-ended, and appeared just like a telegraph tape or a macaroni film. After about one-third of an inch had protruded, it broke off by its own weight, and fell heavily to the base of the bladder.

Turning the light on to the base I saw a small heap of similar ribbons of white material. These were sucked out of the bladder, and seen to be merely tape-like threads of inspissated pus $\frac{1}{8}$ in. broad and $\frac{1}{16}$ in. thick. They were evidently being forced out of an abscess sac into the bladder through a very small channel of communication; and probably the severe pain which the lady had been suffering from for months, and which had necessitated the daily exhibition of large doses of morphia, was caused by the pus being pent up under great pressure.

On introducing a probe into the bladder together with the electric cystoscope I was able to direct it against the opening whence the pus was issuing, and was lucky enough to engage the orifice and push it on into the abscess sac. Immediately a cloud of pus swept over the field and obscured the view. Giving the probe to my assistant, I cautiously raised the pelvis and proceeded to perform laparotomy, expecting to find a suppurating ovary on the right side. After separating a few omental adhesions, I lifted the small gut out of the pelvic cavity and felt for the abscess, but without success. The right ovary felt normal. There was no collection of pus in the peritoneal cavity, for I turned on the searchlight and swept the floor of the pelvis with it. The peritoneum was discoloured and thick on the right side, as it appears when suppuration has been proceeding beneath it. Obviously the abscess was beneath the peritoneum and in the cellular tissue, on the right side of the bladder. I gave up the search and closed the wound, but determined to attack the abscess sac from the vagina, and drain it from below. I made a free incision along the right side of the roof of the vagina on to the probe, expecting to hit the abscess sac without opening the bladder. To my dismay, I found that the probe had slipped out of the sac during the varied manipulations to which the patient had been subjected. After an ineffectual attempt to re-insert it through the narrow orifice by aid of the cystoscope, I turned once

more to the incision in the roof of the vagina, and cautiously dissected back between the bladder base, the rectum, and the uterus, hunting for the emptied abscess sac.

Failing to find it, however, although I had gone dangerously near the right internal iliac vessels, I inserted a drainage tube into the gaping wound in the roof of the vagina, and packed it round with cyanide gauze, hoping almost against hope that the stirring up which I had given to the cellular tissue would act beneficially upon the abscess and induce it to heal. The pus disappeared from the urine, the pain gradually lessened, frequency of micturition diminished, and I learnt some months ago that the patient had quite recovered her health, and that no trace of her bladder trouble existed.

I may incidentally remark that abscesses in communication with the bladder are readily dealt with if the channel of communication is sufficiently large to be felt with the point of the finger, for a pair of dressing forceps can be guided through the dilated urethra into the orifice, and the opening enlarged; but in this case I felt it was hopeless to attempt to feel for an opening situated so deeply between the swollen folds of the mucous membrane and so minute as this was.

Illustrative Prostatic Cases.

I attempted some years ago to scoop out a prostatic collection of crude tubercle by dissecting between the rectum and the prostate from the perineum, but could not reach the gland on account of the intimate adhesions between it and the rectum. The relief was most striking; the patient lost his distressing irritability and pain in a great measure, but, of course, the improvement was temporary, and I believe he died two years after.

The relief experienced was due, I submit, to the drainage of the cellular tissue around the base of the bladder.

Perhaps the most important and convincing case I can lay before you concerns one of our profession.

A powerfully-built man of 53, who had held an important position in Africa with éclat, came to me for the relief of a chronic prostatitis which had troubled him for some years, but which during the last two years had evoked a curious train of neurasthenic symptoms. If he sat down he felt before very long a tickling in the vicinity of the prostate, which at first was slight, but which gradually increased, until it became transformed into a horrible and distressing feeling of stabbing and itching, which spread all over his genitals, and forced him to scratch these parts with violence. This wave sometimes burst upon him with such intensity, and the scratching had to be so severely carried out, that an orgasm like that of coition supervened, no emission, however, taking place. To avoid these diabolical feelings, as he termed them, he was reduced to painting the parts constantly with a 25 per cent. solution of cocaine, to sitting on his hip, or well back on the sacrum, and even this analgesic and these positions were often unavailing. His condition was most pitiable. His genitals were scored with finger-nail marks and his mental despondency was desperate. Most forms of treatment had been attempted and had failed. I discovered a stricture of large calibre, and divided it, but with only temporary relief. After a year's patient trial of known remedies, including counter-irritation with iodine, I made a transverse perineal incision in front of the anus, and dissected between the prostate and the rectum, and drained. He has been perfectly relieved by this procedure, and I saw him last week the picture of mental and bodily health.

Indications. I would suggest, in obstinate inflammatory conditions of the urinary organs of a subacute type, which have resisted ordinary methods of treatment, that an incision may be made over the offending viscus and the cellular tissue in their immediate neighbourhood freely opened with a carefully aseptised finger, the wound being subsequently drained from a week to a fortnight.

Dangers. The aseptic surgeon has entirely lost his fear of the cellular tissue, as he has of the serous cavities, and the parts around the bladder and kidney are nowadays explored freely and without danger. I have myself explored the kidney region over fifty times, and the suprapubic region over sixty times, in performing various operations, and I can only record one case in each section in which the cellular tissue was dangerously affected by leakage from the diseased organ, a condition which would not, of course, occur, if the organ is left untouched as it is by the procedure I advise.

On the other hand, it is difficult to conceive anything more detrimental than a cellulitis set up by a slovenly septic surgeon. Nor is it easy to

realise the damage which may be caused by a surgeon breaking into a tubercular or carcinomatous deposit and permitting the infecting contents to spread themselves over the area which has been laid open merely for drainage.

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NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Tuesday, December 18th, 1894.

Chronic Papillary Patches on the Legs.

A married woman, aged 44, presented the same condition of hard, dry papillary patches on the legs as was demonstrated in the case of a man in the previous week. They had been present, she said, for nine years, and were attended by much irritation. She considered that they had been caused by eating fish, and said that she had at one time had an outbreak of a red eruption all over the body (? urticaria). An exceptional feature in the case was that a group of these hard, dry papules occurred on the calf of the leg. They were all very pruriginous, but the patient had no skin disease elsewhere.

Comments.—In the case last week we referred this peculiar disease to a combination of causes, and I need not now repeat what was then said. As regards the suggestion that these patches are a sort of remainder from an attack of lichen planus, we have in this instance a fact which was not present in our last week's case—that the patient does describe a former attack of a general eruption. It seems, however, more probable that it was an outbreak of urticaria than of lichen planus. You will have observed that, as in our former case, the patient is in excellent health. We may explain her belief that the eruption is due to taking fish by the supposition that she is one of those in whom fish causes pruriginous irritation. In many persons all kinds of fish do this, and one of the first rules in the treatment of all forms of prurigo is to absolutely forbid fish.

Tubercular Ulcer (Lupus necrogenicus) on Arm, possibly from Inoculation.

A man, aged 54, a farm bailiff, apparently in excellent health, presented a sore the size of a half-crown on one forearm. It had been present fourteen months, and was gradually spreading. It was covered by a dry adherent scab, into the under surface of which hypertrophied papillæ projected. On removing this crust the papillæ were torn and bled. The case had been sent by Mr. Hitchens from St. John's Hospital, with the diagnosis of "lupus necrogenicus." The man gave no history of tubercle in the family, and attributed the sore to his having pricked his arm with a thorn. He was the subject of congenital xerodermia, and said that he had a brother in a precisely similar condition.

Comments.—There can be no doubt that this patch is lupus. The interesting point is, How did the man get it? He is accustomed to act as a butcher on occasion, and it is quite possible that he may have dressed a tuberculous sheep. We may remember, however, that butchers very rarely present us with similar conditions, nor can I think that in any large number of cases of lupus there is any great probability that the disease is caused by inoculation. Without, in this instance, venturing any definite opinion, I am quite prepared to believe that any mechanical injury, in a person predisposed, may originate lupus. Whatever may have been the mode of its production, however, we may note that the patch has remained strictly local, and that it is almost wholly unattended by inflammation. It is remarkably dry and quiet, and we may suggest that these conditions are, perhaps, in part, due to the patient's xerodermia. The treatment indicated is, of course, to destroy the patch utterly with caustics.

Smooth Elephantiasis — Recurrent Erysipelas—Cure of a Syphilitic form of Lupus erythematosus.

A man, aged 61, was brought by Mr. Hopkins from the Cleveland Street Sick Asylum. He was the subject of two wholly distinct and somewhat rare maladies. His right foot and leg were in a condition of smooth elephantiasis or solid œdema without papillary growth. He had had numberless attacks of erysipelas in the leg. They had recurred, he said, sometimes as frequently as once a week, and each attack left the leg more swollen. His other ailment consisted in extensive superficial scars on his nose and face, arranged exactly like the bat's wings of lupus erythematosus. The

scars were perfectly sound and white. On his forehead was a large patch of dusky erythema, ill-margined. He had suffered from syphilis in early life.

Comments.—As regards the elephantoid leg there is not much to be said. It is the old story of an injury followed by erysipelas and then of repeatedly recurring attacks of the latter, ending in solid œdema and tissue-hypertrophy. We may allow it to again impress the lesson, so frequently illustrated by similar cases, that the germ poison of erysipelas may remain for years in the part of which it has taken possession, and that it will manifest its activity by periodic outbreaks, with intervals of quiet. The man has told us that he always feels feverish and ill when the erysipelas is on him. As regards his face, I think we may diagnose a syphilitic simulation of lupus erythematosus. Mr. Hopkins tells us that specifics have always been useful, and the cure appears to have been much more easy than it usually is with lupus erythematosus of the scrofulous form. We may note that the old man appears now to be in excellent health, and that his syphilis was very long ago.

A case of severe Iodide Eruption.

In another case brought by Mr. Hopkins from the Cleveland Street Sick Asylum, the patient was a miserable-looking man, covered with extensive ulcerations on his neck, face, scalp and upper extremities. The first suggestion was that he was the subject of granuloma fungoides, for many of the ulcers had more or less tuberous edges. The short duration of the case, however, negatived this supposition, for the man said that his skin had been perfectly clear until ten weeks ago. His statement was that he had worked as a pavior up to three months ago, when he was invalided by an attack of gout, and went into a workhouse. It was during the treatment for gout that the sores on the skin began to form.

Comments.—The case differs from one of ordinary iodide eruption in the fact that there are very few and only ill-marked tubers or papules. The conditions are rather those of extensive ulceration, which in many places has produced scars. Thus, both lower eyelids have been everted. There are, however, if we look for them, here and there some small tubers, just like those so common in the worst cases of iodide poisoning, and many of the ulcers have more or less bossy margins. Unless

the case is one of iodide eruption I do not know what to consider it. The patient has only to-day come under Mr. Hopkins' care, and we have no information as to what drugs have been previously used. The diagnosis which I have ventured is, therefore, subject to correction from the information which will probably be before us next week. It is, however, of great importance to make it promptly, and to act upon it, for the man's life is obviously in danger. The museum contains several portraits of iodide cases which ended fatally. Amongst them is one not wholly unlike the condition of our patient. In that it was attended by ulceration of all the papules and tubers. In that case the man very nearly died before the diagnosis was made. He recovered quickly and completely when the iodide was left off.

Cases of Inoculation of Tubercle by Tattooing.

A very interesting series of cases was brought by Dr. John Murray of Tottenham, which probably illustrated the inoculation of tuberculosis in tattooing. Two brothers, aged 13 and 11, and one of their friends, aged 15, were the subjects. They had all been tattooed on their arms by a lad, aged 17, a brother of two of the patients, who was at the time in an advanced condition of phthisis; he died, in fact, two or three days after the completion of his work on one of the boys. In mixing his pigments he had used his own saliva. The results had been very much the same in the three boys. In some parts the tattooing had been effected without any irritation, whilst in others inflammation had resulted, attended with swelling and the formation of little pustules. No very definite ulceration had taken place. Two of the boys were brought for demonstration, but Dr. Murray stated that in a third the conditions were exactly similar. In all there had been painful swelling of the axillary glands. An interval of nearly two months had elapsed since the tattooing.

Comments. — We are much indebted to Dr. Murray for the trouble he has taken in bringing before us these boys, and for his careful collection of evidence respecting them. I fear that there can be no doubt that a tubercular inoculation has really been effected. Allowing for difference of age and conditions of nutrition of the skin, the patches which have been produced may be said to be not very unlike that in the man whom we have

just seen with lupus of the forearm. We may note that no deep ulceration has been produced, but simply an inflamed patch of swollen skin.

Tuesday, January 8th, 1895.

Multiple Tubercular Sores on all the Limbs, possibly an Early Stage of Lupus Vulgaris—Bazin's Legs.

A fair-complexioned, intelligent girl, aged 9, sent from the London Hospital. She had ulcers on the backs of both hands, near to which were patches of glossy, thickened scar. On her legs there were several ulcers, some of them near to and above the knees, covered by thick, rupial-like crusts. One of the ulcers, near to the ankle, was as large as a shilling, and presented steep borders, as if punched out. She had very sore nostrils.

Comments.—At first sight we should certainly take these lesions to be those of syphilis. The ulcer of the leg is exactly what we used to imagine characteristic of syphilis. So also are the rupial crusts. Those on the hands are much less so, and present conditions resembling lupus. I do not believe that the child is syphilitic. There is nothing in her physiognomy to suggest it, and since we have become familiar with Bazin's legs we have fully recognized that there is nothing distinctive in the characters of the ulcers which are here shown. The multiplicity of the lesions proves that there is some infective element at work, and I should feel but little doubt that it is of a kind allied to tuberculosis. By this I mean that it belongs to the lupus family, as members of which we claim all truly scrofulous affections of the skin itself. In the earliest stage of lupus, when it is developed in multiplicity in young persons, it does not display its distinctive features. In a drawing which we have in the museum, the conditions were at first much like rupia, but subsequently settled down into those typical of lupus vulgaris. I think it very probable that such will be the course of events in this case. We have often had occasion to advert to the curious and very important fact that when lupus is multiple its multiplicity is always attained at a very early stage, and with, at first, such excess of inflammatory action as may conceal the nature of the disease. Of course, I do not regard the ulcer on the leg, deep and punched out as it is, as lupus. It belongs to Bazin's group; but after all the latter is a strumous disease, and there is no reason why we should not meet with it in company with lupus.

Syphilis in a Mother and Child, with peculiar features in the latter.

A mother and infant were brought by Mr. Hitchens. The mother had had a sore inside her lips for three months, with swelling of the glands under the jaw. It had been treated as syphilis. The child, now a year old, had been quite well until two months ago, when it had become the subject of very sore lips; had large patches form in the palms of its hands, and a suppurating onychia of its right great toe. The patches in the palms of the hands were very peculiar, being considerably raised and somewhat papillary.

Comments.—The child's condition is somewhat unusual. It presents none of the more ordinary indications of congenital syphilis, and the date at which its symptoms began fits well with the idea that it has contracted primary syphilis from its mother. It is possible that the chancre may have been on its lip, although there is no clear proof of this.

A doubtful example of the Crateriform Ulcer.

A woman, aged 49, sent by Dr. Vinrace, presented a very peculiar ulcer on her right lower eyelid. It was described as having commenced two months ago as a small boil, and had now become elevated with a crater-like depression in its centre. During the last few weeks a number of other superficial patches, with crusts, had formed on the face near it. The woman had lost the end of her nose and the whole of the septum nasi, but she had no other indications of syphilis. She had suffered from diabetes for more than two years, but was still stout and in fair health. The history given was that the disease which destroyed her nose and vomer had occurred thirteen years ago.

Comments.—I do not think that there can be any doubt from the way in which the end of the nose and its septum have been destroyed that this patient has suffered from syphilis. The character of the scar is quite distinctive from that of lupus vulgaris. There does not, however, seem to be any reason to believe that her present symptoms are specific. The ulcer on the eyelid looks very like what I have described under the name of the crateriform ulcer. This ulcer is a peculiar form of epithelial cancer, which develops very rapidly and breaks down in the middle to form a deep crater. It is altogether different in its clinical course and

external features to any other form of malignant disease of the skin. A very important point about it is that, however formidable it may look, if it be freely excised there appears to be but little risk of recurrence.

Congenital Xerodermia, with history of family prevalence in three generations.

A man, aged 32, the subject of xerodermia, who was brought by Dr. David Walsh, gave an interesting family history. His father, grandfather, and three uncles had been the subjects of the same malady, and one of his brothers, four of his sisters, and five cousins also suffered. It was believed to have been more severe in the earlier generations than in the present one. In the patient it was a slight form, but universal with the exception of the face. Dr. Walsh had been giving thyroid tablets for about three months, and both he and the patient were well satisfied with the result, the skin being softer than formerly. The patient stated that he had great difficulty in perspiration.

Case of very extensive Multiple Lupus.

The subject of this case was a boy, now aged 10 years, whose case has been repeatedly published, and of whom several portraits have been taken illustrating different stages of his malady. These portraits were produced and placed by the side of the patient for comparison with his present condition. He was now covered with enormous patches of lupus vulgaris in the dry exfoliative stage. The patches were so large that a good deal more than half of the surface of the limbs and trunk was occupied by them, and the greater part of his scalp was also covered. The patches, although occurring on all parts, were not arranged symmetrically.

Comments.—This case is by far the most extensive development of lupus vulgaris that I have ever seen. It began, soon after the boy's vaccination, as a general eruption which was pustular, lichenoid and in parts almost rupial. In the course of about a year it had developed the characters of lupus vulgaris, and these it has since kept, its patches having become quite quiet and slowly spreading at their edges. As the portraits show, they have increased during the last six years to many times their former dimensions. An exceptional feature is that the lupus has spread almost all over the boy's scalp, destroying his hair. We often see the scalp affected in lupus erythematosus, but some authorities tell us that it never suffers from lupus vulgaris. This is a mistake. I have repeatedly seen it spread on to the scalp, and when it does so the conditions assumed are indistinguishable from lupus erythematosus. This is one of the facts to which I appeal in proof that these maladies are closely allied.

A CASE OF

ENTERIC FEVER TREATED BY THE CONTINUOUS BATH.

BY

FRED. J. SMITH, M.D., M.R.O.P.,
Assistant-Physician to the London Hospital.

By the side of Dr. Barr's number of cases treated by continuous immersion, this solitary case may appear uninteresting, but the effects of the bath were so marked, and the symptoms before treatment so severe, that I think the case, even alone, is worth putting on record.

The patient was a girl, aged 18, who was first taken ill, apparently, on the 6th of September, 1894, but it was not till the 17th of September that I saw her in consultation with Mr. Hamlin, and, recognizing the critical nature of the case, and the unsuitability of her surroundings for nursing, etc., advised her immediate removal to the London Hospital. On admission, her condition was noted as follows:—has been semi-conscious and delirious for twenty-four hours; rash and other features point to typhoid fever, probably late in the second week; temperature, 104.6°; pulse, 140, fair tone and volume; no evidence of any active complications in lungs or elsewhere.

Tepid sponging, with ice cradling in the intervals was at once adopted, with good result, but only for a few hours, and by the afternoon of the 18th the temperature was 105°, and the patient was comatose, and had also had slight hæmorrhage from the bowel. In the evening of the 18th she was placed in a bath at 90°, rapidly reduced to 65°; this reduced the temperature to 102.6° (rectal), and the pulse from 140 to 112. Her

general condition had also improved greatly, but all improvement was only temporary, and by the afternoon of the 19th the temperature was 105° in the axilla, and the comatose condition had returned.

Seeing the good (temporary) effects of cold bathing, I now determined to adopt the plan recommended by Dr. Barr, and, after considerable trouble, arrangements were made. I cannot pass this incident over without mentioning, in terms of the highest praise, the ready assistance given to me by my house physician, Dr. H. L. Rutter, and by Sister Rachel and the nurses. The practical difficulties to be overcome were very great, and had not these all worked hard and willingly, could not have been surmounted.

A long bath was fitted up in the ward bath-room, with a kind of hammock, in which the patient could lie comfortably, covered with a blanket, and supported by pillows. With some trouble, the temperature of the water was maintained between 93° and 96°. The effect was as marked as it had been previously, in an hour's time the temperature had been reduced two degrees, and the pulse from 150 to 112. She was regaining consciousness, and took food without difficulty. She slept well in the bath during the night. On the morning of the 22nd, having been continuously immersed from the evening of the 19th, the patient was taken out of the bath at 11 for the purpose of cleaning it; the temperature was then 103.4° in the rectum, and it steadily rose to 105.4°, till she was replaced in the water. With the rise in the temperature all the bad symptoms, coma, etc., began to return.

From this time the patient was removed every day for a couple of hours while the bath was being cleaned, and an enema given. Invariably this removal resulted in a steady rise of temperature, some two or three degrees. On the 24th, Dr. Rutter reported to me that there were signs of pneumonia at the right base, but this did not seem to me sufficient to cause me to omit the bath. On October 11th an abscess was opened over the sacrum; no dressing was applied; it simply drained into the bath. On October 22nd she was finally removed from the bath, as the pyrexia was not excessive when out of it for an hour. On November 1st her temperature became normal, and remained so. She left the hospital quite well early in December.

Remarks. It certainly requires personal ac-

quaintance with a patient before and during the bath treatment to enable one to fully realize the marked difference that rapidly ensues. I have not the slightest doubt that had not the treatment been adopted here the patient must have succumbed, an opinion in which all who saw her fully concurred. She was comatose, and I might say actually moribund, when placed in the water, and within a few hours regained consciousness, and seemed, as it were, temporarily out of danger. This happened not once, but several times. From a therapeutical point of view, then, continuous immersion requires no further discussion; but there are one or two other matters worth a little consideration.

The temperature of the water. In the light of this case I am led to believe that with a water temperature somewhere between 93° and 96°, we are able to get all the anti-pyretic action that is likely to be required. It is a temperature which is comfortable and comforting to the patient (my patient always begged to be replaced in the bath when temporarily removed), and, in my opinion, the one least likely to do harm from long continuance.

Complications under treatment. Pneumonia seems to have definitely occurred (I did not actually listen to the lung myself, but have no reasonable doubt) in this case, but if influenced at all by the bath, was so in a favourable direction. I could find no reason for ascribing its onset to the treatment adopted. A large abscess formed over the sacrum. This is, of course, a complication always liable to occur in any case of typhoid fever, and I attribute the happy manner of its healing in no small degree to the continuous bath. No troublesome dressing was required, and pressure to any undue extent can be more easily avoided than in an ordinary bed.

Action of the remedy. It is not my purpose to enter into any discussion on this point from the experience of one case only, but I was very forcibly struck by the purple congestion of the face (the only part not in the water), and was thus led to support the view that probably a determination of blood to the kidney and consequent diuresis has a good deal to do with the beneficial effects.

Drugs while in the bath. It is my custom to treat typhoid cases nearly throughout with small doses (gr.j or gr.ss) of calomel every night, and with salol gr.x three times a day, but in this case I

am bound to admit that I think they contributed nothing to the cure.

Adaptability to private cases. After the experience of the difficulties, while not underrating them, I am inclined to think that with a little trouble the method might be tried in any middle-class house where a large bath can be obtained, and certainly in any house where a good bathroom exists with a hot and cold water supply.

Dangers attendant on the treatment. These are, I believe, non-existent from the patient's point of view. The only one I see is to the nurses from splashing of the water, and this, with care, may be quite avoided, and, at most, is not greater than that incurred by them in sponging a case of enteric fever. The pollution of such a large bulk of water is hygienically a more serious matter, but should be no bar to the employment of the bath with reasonable precautions.

THERAPEUTICAL NOTES AND FORMULÆ.

On the Action of Chloral Hydrate upon the Kidneys.—The knowledge possessed hitherto as to the effects of chloral upon the kidneys has been rather scanty. The author, in a series of experiments upon animals, has shown that, when administered by intraperitoneal injections, this substance, after one or but a few injections, determines an irritation of the functional epithelial cells of the kidneys, shown by a distinct fatty-granular degeneration of the epithelium of the convoluted tubes; after a more prolonged use this degeneration implicates all the tubules with the exception of the straight ones. In more severe cases there is a granular swelling of all the renal epithelium, showing all the characteristics of a beginning acute parenchymatous nephritis. The malpighian tufts, however, always appear to remain normal, and there never is any implication of the interstitial connective tissue. When administered by the stomach, the same lesions occur, but in a slighter form, and only after prolonged use. These renal lesions, unless too far advanced, commonly disappear after the withdrawal of the drug. When far advanced it is difficult to say whether these lesions are still curable, for in any individual case it is impossible to diagnose their presence, since, even in cases in which autopsy revealed the most severe lesions, albumen had never been pre-

sent. Among other cases the author was enabled to observe a patient in whom autopsy showed most marked renal change. The woman in question had taken two to three grammes of the drug every evening for fourteen months, and yet her urine never showed the presence of albumin. This very interesting fact explains why we have hitherto heard so little of the bad effects of chloral upon the kidneys.

This study evidently shows that the greatest care is necessary in the administration of chloral to people suffering from renal troubles.

(*Gazz. degli Ospea.*)

Exalgine.—Accidents having been noticed to follow the administration of single doses of 50 centigrammes of exalgine (about 8 grains), M. Dujardin-Beaumetz reported to the Academie de Medecine that the drug must be carefully guarded in use, and a single dose of over 25 centigrammes (about 4 grains) must be avoided.

(*Repertoire de Pharmacie.*)

Chloasma of Pregnancy.—

℞	Zinci oxidi	...	...	gr.vj
	Hydrarg. ammon.		...	gr.ij
	Ol. theobrom.	...	...	ʒv
	Ol. rici.	...	...	ʒv
	Ess. ros.	...	...	gtt.xx

M. Sig.: Apply to the face night and morning.

(HARE, *Coll. and Clin. Rec.*)

Application for Warts.—Kaposi gives the following:—

℞	Flowers of sulphur	...	20 parts
	Glycerine	...	... 50 parts
	Pure acetic acid	...	10 parts

M. This is to be painted on daily for several days.

(*Revue de Therap. Med. Chirurg.*)

A Formula for Bromoform.—W. Lyon recommends the following:—

℞	Bromoform	...	...	16 parts
	Alcohol	...	...	7 parts
	Glycerine	...	...	35 parts
	Tincture of cardamoms			7 parts

Another way to administer the drug is a solution with fatty oils or as an emulsion with gum.

(*Rep. de Pharm.*)

A CLINICAL LECTURE

ON

ASCITES—CIRRHOSIS OF LIVER.

Delivered at Charing Cross Hospital, Jan. 7, 1895,

By T. HENRY GREEN, M.D., F.R.C.P.,

Physician to the Hospital, and to the Hospital for
Consumption, Brompton.

GENTLEMEN,—This patient, who was admitted about a week ago, is 37 years of age. She came here on account of swelling of her abdomen, telling us that she first noticed it about a fortnight before her admission.

In the first place, let us examine the abdomen. It is always wise in practice to direct one's attention first to the obtrusive symptom. The symmetrical enlargement of the abdomen, slightly bulging in the flanks, the prominence of the umbilicus, that fluctuation on percussion, together with the alteration in the level of the fluid as we change the position of the patient, leave no doubt as to the existence of ascites. The recognition of this presents no difficulty; but I would here remark that when the patient is a woman, it is well to bear in mind the possibility of ovarian dropsy, as this may sometimes rather closely simulate ascites, especially if, as is sometimes the case, the encysted is associated with free ascitic fluid. There is, however, no such question here, so that this elementary part of the diagnosis need not detain us.

A much more important point, of course, is the cause of the ascites—of what disease is the ascites symptomatic? The answer to this question is often easy, but occasionally it is difficult, and it may be impossible to speak with any degree of certainty. There is, I think, no difficulty in the case before us; but in order to avoid error, it is well to be guided by methods and principles in diagnosis, and I would advise you in all cases to endeavour in the first place to determine to which of three following groups the case belongs: (1) Ascites, as part of general dropsy, due to cardiac, pulmonary, or renal disease; (2) ascites from

chronic disease of the peritoneum; (3) ascites from obstructed portal circulation.

(1) In the first group of cases—those in which the ascites is part of general dropsy—the discovery of valvular disease of the heart, of emphysema, or of renal disease will usually be sufficient for diagnosis. But remember in old cardiac cases the liver may become fibroid, the ascites be excessive, and persist after all other dropsy has disappeared. This is exemplified by the girl in adjoining bed. Ascites out of proportion to the rest of the dropsy points to a local cause.

(2) Ascites from chronic disease of the peritoneum often presents some difficulty. We discussed such a case on a former occasion. The two most important physical indications of such disease are a want of the natural smoothness and suppleness in the feel of the abdomen, due to matting together of the intestines, and dulness at the umbilicus, due to a shortened mesentery, preventing the intestines floating on the top of the fluid. Tubercle is much the most frequent cause of this condition—then disseminated carcinoma.

(3) The third and much the largest group of cases is that in which the ascites is due to some local obstruction of the portal circulation. It is to this group that the case before us obviously belongs—the ascites is the only dropsy. There is no discoverable disease of heart or kidneys, and nothing in the physical examination of the abdomen or in the symptoms to justify a suspicion of disease of the peritoneum.

In the next place we must consider the causes of obstruction to the portal circulation. We will briefly allude to them, because it is always wise to keep them in mind when attempting to make a diagnosis. In the majority of cases obstruction to the portal circulation is due to disease of the liver; much less frequently to some obstruction of the portal vein, which may be quite independent of hepatic disease.

Let us consider the least common class of cases first—portal obstruction which is not due to liver disease. The most frequent cause here is some obstruction to the portal vein in the hilus of the liver, usually malignant growth. Malignant disease of the stomach, or pancreas, may thus

involve the hilus of the liver; and growths originating in the liver itself may obviously do so likewise. But the disease which implicates the portal vein nearly always implicates also the common bile-duct, so that obstructive jaundice is met with in addition to ascites. It is the association of these two symptoms that points to the portal vein being obstructed before it enters the liver. In our patient there is no jaundice, and the age almost excludes carcinoma. Thrombosis of the portal vein, and obstruction of the hepatic vein just as it enters the inferior vena cava—possible causes of portal obstruction—are so rare, that we may practically exclude them from our diagnosis.

Portal obstruction due to disease of the liver is most frequently due to some *fibroid* change in the organ. It matters not whether this change is inter-lobular, peri-lobular, or inter-cellular; all such changes tend to obstruct the portal circulation in a greater or less degree, and thus to cause ascites. Of the etiological factors here, *alcohol* stands pre-eminent. Next in frequency is *long-continued mechanical congestion*, such as results from heart disease, of which we have a typical example in the next bed. You know how often in a case of heart disease, if it has lasted long, the liver becomes fibroid and produces ascites, and this may persist when the general dropsy has disappeared. The third cause of fibroid liver is *syphilis*. With regard to this, we must be guided principally by any other evidence of this disease, and by the absence of evidence as to abuse of alcohol. A thickening of the capsule of the liver, a *peri-hepatitis*, may sometimes so squeeze and distort the organ as to obstruct the circulation. But this is usually due to syphilis or alcohol, and in my experience is rarely met with.

Of other diseases of the liver that obstruct the portal circulation there remains only *infiltrated carcinoma*. This may cause ascites, but the great increase in the size of the organ, the usual association of jaundice, together with the age. of the patient, will usually suffice for diagnosis.

To return to our patient. The ascites, we have already concluded, is due to portal obstruction, and in the absence of jaundice it is probable that the cause of this is in the liver itself, and not in the hilus. This being so, we ought to look for any evidence of liver disease in addition to the ascites. And if we could discover that the organ was *too large*, or *too small*, or *too hard*, we should then

have very important additional evidence of the existence of disease. But if we try to feel the liver —and, remember, always palpate before you percuss—owing to the large amount of fluid, we find nothing at all; even by suddenly depressing the hand so as to displace the fluid, and turning the patient on the left side, the result is the same—a negative one. Feeling the liver affords the most valuable indication of a fibroid change in it. A hard and rounded edge is the most important physical sign of cirrhosis, especially in the early stage, before the development of ascites.

An attempt to ascertain the size of the liver may be made by percussion, but where the ascites is considerable, owing to the displacement upwards and tilting of the lower edge of the organ, the results of percussion are fallacious. In fine, when there is much ascites, you must not expect to learn much from a physical examination of the liver, although you may do so after the fluid has been removed.

Failing to get much help from an examination of the liver, let us look for evidences of portal obstruction other than the ascites, and for any evidence of the abuse of alcohol. The earliest symptoms of obstructed portal circulation are usually gastro-intestinal ones; one of the most important is a tendency to diarrhœa. This often precedes, by a considerable period, development of the ascites. You will notice in this patient's history that during the last twelve months she has usually had one or two stools in twenty-four hours, and that the stools have been loose. It is the looseness, and not the increased frequency, that indicates the existence of portal obstruction and overfulness of the portal vessels. Gastric symptoms have a similar diagnostic import. The obstructed portal circulation causes catarrh of the stomach, resulting in more or less impairment of digestion, with a frequent tendency to nausea, and especially morning sickness. These symptoms our patient has suffered from for several months past.

Evidences of the abuse of alcohol are numerous. The patient admits beer and spirits; she is liable to "nervousness and trembling"; sleep is disturbed by dreams; and on several occasions she has brought up some blood with·her morning cough. Alcoholics are prone to bleed from nose and pharynx. There has been no hæmatemesis. All these facts, coupled with the patient's symptoms, point to the abuse of alcohol. In many of these

cases it is difficult to estimate the extent of the abuse, but it is always wise to remember that what is one man's food is another man's poison. Very small quantities of alcohol are quite sufficient to produce, in some people, definite morbid change.

I would here repeat what I have so often told you before, that it is impossible in these cases to draw any sharp line of demarcation between the symptoms due to the direct effect of the alcohol on the stomach, and those due to obstructed portal circulation. You know that the effect of alcohol on the stomach is to produce gastric catarrh, causing some degree of morning sickness and impairment of digestion. Such symptoms, usually with a tendency to diarrhœa, commonly precede for a considerable period all reliable evidences of the existence of cirrhosis. With the development of the hepatic lesion the circulation is interfered with, and these gastro-intestinal symptoms are intensified.

To recapitulate, our diagnosis runs thus :—The ascites is due to portal obstruction, the portal obstruction is hepatic, and the disease of the liver is probably alcoholic cirrhosis. After the abdomen has been tapped, we shall see how far our examination of the liver confirms this.

Having arrived at this conclusion, one next asks the question, What form of cirrhosis? We are now taught that there are several varieties of cirrhosis of the liver—atrophic, hypertrophic, biliary, and so on. Doubtless, by far the most common is that which is usually called *atrophic* cirrhosis, in which, with the progress of the disease, the liver gradually diminishes, until it may not be more than half its natural size. I would ask you to note here that probably too much has been said about diminution in the size of these livers. Certainly, so far as my own experience goes, diminution in size is much less frequent than is stated. In a large number of cases you will find, on post-mortem examination, that the liver in cirrhosis is *not* smaller than natural ; not infrequently it is somewhat larger. So be careful that you do not make too much of the shrinking of the organ, and do not make the mistake—a not uncommon one—of concluding that, because the liver is not diminished in size, the patient is in an early stage of the disease. In *hypertrophic* cirrhosis the liver is considerably increased in size, the surface is smooth, there is usually not much ascites (often none at all), and

jaundice is a prominent symptom. This, obviously, is not the condition here. There is no jaundice and no evidence of enlargement of the liver. A large liver with cirrhosis is sometimes due to infiltration of the cells of the liver with fat —fatty infiltration.

Our patient, therefore, is probably suffering from the common form of alcoholic cirrhosis, the atrophic, and at present she seems to complain of little except the distension of her abdomen. She is taking her food fairly well, and it is mainly the ascites which causes her trouble. The ascites itself, quite apart from its cause, gives rise to more or less definite symptoms. The distension of the abdomen causes discomfort, the fluid pushes up the diaphragm and embarrasses the heart and respiration. The movements of the stomach and intestines are interfered with, and thus gastric digestion is still further impaired ; and diminished peristalsis often causes constipation, when previously there was diarrhœa. Pressure on the iliac veins leads to distension of the superficial abdominal veins, and to swelling of the lower extremities ; and pressure on the renal veins causes congestion of the kidneys and diminution in the quantity of urine. This patient's urine is scanty, concentrated, with large deposit of urates.

I will now ask you to consider with me for a few moments what is the natural progress of a case of this kind. Remember we can relieve, or we may even cure, the ascites, but we cannot materially influence the progressive atrophy of the liver. True, by cutting off the alcohol we may retard the fibrosis, and in the *earlier* stages this is all-important, but when this change is at all advanced *progressive* destruction of liver cells is almost certain. The symptoms of cirrhosis are by no means confined to those due to obstruction of the portal circulation. With the destruction of the hepatic cells the functions of the organ are progressively impaired, and food metabolism is markedly interfered with. This accounts, to a very great extent, for the progressive emaciation and failure of health and strength which characterise the advanced stages of cirrhosis. We are very apt to forget this when making our forecast, and in estimating the possibilities of treatment. The forecast in the early stage of alcoholic cirrhosis is very different from that in the later one. In the early stage—before ascites and before hæmatemesis—complete recovery is possible,

provided you can cut off the alcohol. But in the great majority of cases in which the disease is so advanced as to have led to hæmatemesis or ascites, death is (as far as my experience goes) rarely delayed beyond two years, and it is usually much sooner. This is all-important to remember.

Now as to *treatment.* I have said that we may diminish the ascites, perhaps even cure it, and keep our patient free from it for a considerable time, but we cannot do much to influence the fibroid change in the liver, though by stopping alcohol we may perhaps retard its progress. In attempting to influence the ascites, remember that as the portal capillaries become obstructed, the circulation is carried on to a greater or less extent by collateral vessels. There are numerous channels by means of which the portal blood may reach the general circulation without going through the liver. Anastomosis may be sufficient to prevent ascites; and, as you know, in some cases the vessels at the lower end of the œsophagus become so varicose that dangerous hæmatemesis and not ascites is the prominent symptom. Now the ascites, by the pressure it exercises, interferes with this establishment of this collateral circulation. Hence, especially, it calls for treatment. As long as the ascitic fluid does not cause pressure symptoms, it does no harm. Until we have distinct evidence that the fluid is exercising injurious pressure, it is best to leave it alone.

How can we, by treatment, diminish the ascites? In the first place, I would say do not expect to do much by means of drugs. The only efficient way to diminish ascites is by paracentesis. Do not make the mistake we have made before, many of us, of expecting that by drugging the patient we shall get rid of the ascitic fluid. I do not say drugs are useless; far from it; they will help, but they will not be sufficient. There are three kinds of drugs which are likely to be beneficial—purgatives, diuretics, and tonics. Purgatives stand first. Be careful not to purge your patient too much. As a rule I use Carlsbad Salt—one heaped teaspoonful or more in half a pint of hot water the first thing in the morning, just sufficient to produce one or two slightly loose stools. It is usually wise to content yourself with this, so as not to weaken your patient. Diuretics are less useful; squill, digitalis, copaiba, broom, mercury, potash, &c., have been employed. Copaiba is sometimes beneficial, and increases the quantity of urine. I have not seen it do harm, and usually give it (gr. v in pill, four

times daily). In connection with diuretics, remember that as long as the ascitic fluid obstructs the renal circulation by pressure on the renal veins, these remedies will be inert; it is after paracentesis that they become useful.

Tonics should occupy a prominent place in the treatment, especially such as improve appetite and digestion—stomach tonics. Some alkaline bitter, with nux vomica, taken two or three times a day shortly before meals, is usually of much service; acids after food are sometimes indicated. Iron is occasionally, but I think only occasionally, of use. In attempting to explain the undoubted value of tonics, it must be remembered that although the pathology of ascites is imperfect, impaired nutrition of vessels and altered blood are probably concerned in the causation of the dropsy. The dietary should be liberal and varied, due regard being had to the digestive powers of the patient. Liquids should be restricted.

Paracentesis should be performed as soon as the ascites is sufficient to exercise injurious pressure, and perhaps marked diminution in the quantity of urine is the most reliable indication of this. This patient has been in hospital a week, and in spite of the drug treatment I have indicated the urine is only twenty-four ounces, and the abdomen has increased in size. We shall therefore remove the fluid.

As to the method of procedure, you may use the ordinary trocar, or you may evacuate the fluid more gradually by means of Southey's trocar. If the symptoms are not urgent, and there is no hurry, I prefer Southey's trocar. It causes no pain or discomfort, and the fluid drains away gradually while the patient is asleep; twenty-four to forty-eight hours often being occupied in the process. When the abdomen has been drained it is important to remember that, the pressure being removed from the vessels, it will probably fill up again quickly. The abdomen should therefore be well supported by a flannel bandage after the operation.

After the abdomen has been drained, the drug treatment will be continued, and with better prospect of success. We must anticipate the probability that the paracentesis will have to be repeated, and this perhaps several times; and remember, that although in some cases the ascites subsides, and health and strength improve, in too many the patient succumbs either from the original disease or from some intercurrent affection.

A CLINICAL LECTURE

ON

ACQUIRED DEFORMITIES.

Delivered at Guy's Hospital, by

W. ARBUTHNOT LANE, M.S., F.R.C.S.,

Assistant-Surgeon to the Hospital.

GENTLEMEN,—I would urge on you very strongly when you are studying clinically the several surgical conditions with which you must render yourselves familiar, not to be satisfied with accepting the usual text-book statements of their causation, unless, on applying to them the test of your sound common sense, you find that they are perfectly intelligible and rational.

You may, I think, conclude that any explanation of the causation of a condition that, after thoughtful consideration, appears confused, complicated, and verbose, is probably false.

When a writer has in his own mind no definite idea of the cause of a morbid condition, he often takes great pains to render his description of it as unintelligible and vague as possible, or he smothers the reader with a number of views held by various authors, each usually utterly at variance with the other.

General laws are, as a rule, very simple, readily intelligible and capable of proof, and the importance of a principle is generally directly proportional in degree to its simplicity.

I would, therefore, advise you to avoid assimilating in an unchanged form the accumulation of what passes for knowledge, unless you have first digested it, absorbing the reasonable, and excreting anything which cannot bear the closest criticism.

I intend in this lecture to attempt to explain the anatomy and the causation of the several acquired deformities which are painfully familiar to us in the out-patient room, and which too frequently derive very little benefit from our treatment of them, for the reason that we are unable to place our patients under such circumstances as would bring about their recovery.

The deformities I allude to are knock-knee, flat-foot, lateral curvature, and dorsal excurvation. These I will call for convenience "resting" deformities, the reason for which I will show later.

It is obvious that if you are unfamiliar with the anatomy, physiology and causation of these conditions, you are not in a good position to treat them. You are still more unfortunately placed if the idea you have of the mode of their production, etc., is absolutely false. There is, perhaps, no branch of surgery about which so many various theories of causation have been evolved, and about which so much obscurity and ignorance exists even in the best standard works on surgery.

One is very liable to imagine from a knowledge of anatomy as taught by the text-books, that the construction of the human body does not allow of much variation. A little careful study will, however, show you that not only do the forms of the bones of the human adult skeleton vary with such movements as are performed habitually, and with such routine ˉattitudes as are assumed by the vigorous individual, but also do the details of the structure and the functions of the several joints.

If an individual is habitually engaged in performing a certain movement or sequence of movements of activity, the form of the skeleton varies from the normal in a degree which is proportionate to the length of the period during which the movement has been performed, and to the amount of energy expended in the act. During a single performance of the attitude of activity there are present numerous tendencies for the bones and joints to undergo changes in form. Their constant repetition enables the tendencies to become actualities. The earliest variation from the normal consists in the fixation of the physiological attitude which is naturally assumed during the performance of such a movement of activity, while the later changes are exaggerations of the same attitude due to changes in the bones and in the intervening soft structures. In other words, *the peculiar character, of the anatomy of the labourer is, first, the fixation, and subsequently the exaggeration of a normal physiological attitude of activity.*

A workman who for a large number of years has followed the same laborious occupation, possesses an anatomy which is unmistakably characteristic of that occupation, and which in many cases differs from what we are accustomed to regard as the normal anatomy of the human subject, infinitely more than the latter differs from that of the ape.

The variations exist not only in the separate bones and joints, but in the arrangement of these bones.

Compare, for instance, the clavicle and scapula of the shoemaker with those of the sailor and deal porter, and the least observant will see at once the very great difference in the form of the several

bones. A little consideration of the peculiar physiology of each occupation will show you the factors that have produced the characteristic form of each bone. They are chiefly ossification of muscles along the lines of habitual traction, and the alteration in form which results from the habitual transmission of considerable force in certain directions. Another important factor, though perhaps a less obvious one, is the formation of a new bony structure, with a view to the better transmission of force under any mechanical circumstances peculiar to the occupation or acquired physiology of the individual. This is shown very well in the atlas of a man whose function it has been during a long lifetime to sew boots. In this individual, the obliquely-placed head is supported on a column of bone which grows upwards from the lateral mass of the atlas, and forms for itself an articulation with the under surface of the jugular process of the occipital bone. If you wish to pursue the subject further, you will find the anatomy and physiology of the shoemaker fully described in the *Journal of Anatomy and Physiology*, July, 1888.

Then examine the spine of the coal-trimmer, whose anatomy and physiology is described in the
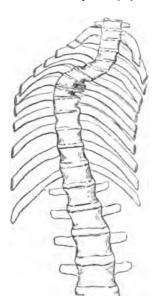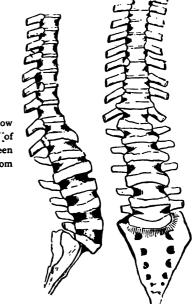
Fig. 1 represents the greater part of the spinal column and the posterior portion of the thorax of a brewer's drayman, whose body I examined in the dissecting room of the hospital.

same Journal (April, 1887), and you will find that in consequence of the forcible rotation of the trunk upon the pelvis in throwing coal to places at a distance, the neural arch of the fourth lumbar vertebra is completely divided, and an arthrodial joint is formed between the bodies of the fourth and fifth vertebræ, the fibro-cartilage which originally

existed there having been more or less completely removed.

Again, observe the spinal column of this drayman, and see the large single lateral curve, with changes

Figs. 2 and 3 show the spinal column of a coal heaver, as seen from the side and from the front.

in the form of the several portions of the vertebræ in the intervening fibro-cartilages, and in arrangement of the vertebræ, ribs, etc.; or look at the spine of the coal-heaver, and notice the obliteration of the dorsal and lumbar curves, the removal of the amphiarthrodial joints between the lumbar vertebræ, with their more or less complete synostosis, together with the downward and forward displace-

Fig. 4 represents the 4th and 5th lumbar vertebræ and the sacrum of the coal heaver in vertical antero-posterior section, and illustrates the form of spondylolisthesis found in this class of labourer.

ment of the fifth lumbar vertebræ off the sacrum, a condition dignified by the name spondylolisthesis. The conditions which result from carrying habitually heavy loads on the head are well shown by figs. 7 and 8.

Many of the changes in the form of the ends of

the bones and in the character of the joints were formerly observed by pathologists, and were by them regarded as examples of a disease called

Fig. 5 shows the form of spondylolisthesis that occurs in such labourers as carry heavy loads on their backs less heavy than those borne by the coal heaver, and who pick them off and replace them on the ground. The arthrodial joint seen between the 5th lumbar vertebra and altered upper piece of the sacrum is produced by this greater freedom of movement.

rheumatoid arthritis, with which, of course, they have no causal connection or relation whatever, though they are in appearance somewhat similar.

Fig. 6 presents a condition the reverse of spondylolisthesis, namely, a displacement of the straightened or even anteriorly concave lumbar spine backward upon the sacrum, and the consequent transmission of a large part of the superjacent weight through enormously developed dense lumbar and sacral spinous processes. This condition results from carrying on the head, or the load being habitually borne in front of the chest or trunk.

The human skeleton retains what is regarded as its normal anatomy only as long as it is called upon to perform normal functions, and those not excessively.
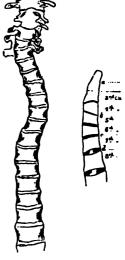
I would now point out to you that, for the so-called normal condition of the skeleton, it is necessary during growing life that the individual shall combine attitudes of activity with attitudes of rest, and that the attitudes of activity as well as those of rest, be varied in character. By an *attitude of rest* is meant a position in which the superjacent weight is transmitted through a joint, or joints, with a minimum of expenditure of muscular energy, the form of the bones and the ligaments sustaining the

chief strain, the muscles in many cases performing only the passive function of a ligament.

As already explained, during the single assumption of an attitude of activity, there exists a tendency to change, both in the forms of the bones and of the joints, resulting in the alteration if the attitude be assumed habitually. So also during the period of a single assumption of an attitude of rest, there

Fig. 7 represents, as seen from the front, the spine of a man, who was employed in carrying on his head loads of some considerable weight.

Fig. 8 represents the upper part of this spine in vertical antero-posterior section, and shows the considerable destruction of the fibro cartilages in the cervical region which results from the habitual transmission of abnormal pressure.

exist tendencies to change, both in the form of the bones and of the joints, resulting in the alteration if the attitude be constantly adopted. This occurs very much more readily and extensively in the growing subject, than in such as are fully developed, for the following reason, which I will state in the form of a general law, the importance of which you will readily perceive.

In the young subject, the rate of growth of any portion of an epiphysial line varies inversely as the amount of pressure it transmits. In other words, if one half of an epiphysial line transmits habitually an amount of pressure which is greater than normal, the amount of bone which it develops is correspondingly less than that normally produced. If, on the contrary, the other half of the epiphysial line is subject habitually to a subnormal pressure, the amount of bone developed by it is proportionately greater than the normal. In all the resting postures, the mechanism of the skeleton is such that one portion of a growing line is exposed to an abnormal amount of pressure, while another portion is exposed to a much less or even subnormal amount of pressure. The frequent assumption of a single attitude of rest, not corrected by suitable variations in the attitudes of activity and rest, results finally in

a progressive alteration in the form and function of the bones and joints. The soft parts intervening between portions of bone which habitually transmit an abnormal amount of pressure, first become thinned and fibrous, and later may be completely removed. If the bones move freely upon one another, the exposed osseous surfaces become dense and eburnated, but if very little movement takes place, the opposing surfaces of bone may unite. It is obvious that when these changes have advanced to any extent, they cannot be obliterated by treatment, the original form of the skeleton being lost for ever. This law has a most important bearing on the evolution and treatment of acquired deformities.

One knows also that the more vigorous and robust the child, the less likely he is to assume attitudes of rest for any time; while the feeble-bodied and frail is but little disposed to expend his scanty muscular power in activity, but prefers to assume such easy postures as make as little demand as possible upon his store of energy, the particular variety of resting posture varying to some extent with the age, surroundings, and often also with the occupation of the individual.

If you accept as true the views which I have attempted to place before you, I will pass on to the consideration of the causation of knock-knee.

If the active erect posture be assumed, and a plumb-line be dropped through the head of the

9   10   11   12
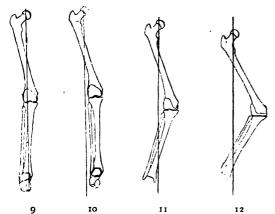
Fig. 9 represents the normal mechanical condition, and figs. 10, 11, and 12 the progressive diminution in the external femoro-tibial angle.
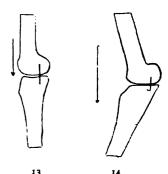
femur, it will be seen to fall much outside the centre of the joint. If the easy erect posture be assumed, and the weight of the body be supported upon one leg, the total weight of the body falls upon the knee-joint through a point in a transverse

vertical plane, which is still more external to the centre of the joint. If this posture be habitually adopted, the outer portion of the growing lines of the femur and tibia form bone more slowly than normal, while their inner segments form it more rapidly. Consequently the internal condyle becomes longer, and the outer shorter than in the normal skeleton. As the external femoro-tibial angle diminishes, the patella is displaced further and further outwards, till finally it rests on the outer surface of the external condyle in the most advanced condition of knock-knee.

Associated with "knock-knee," but sometimes arising independently of it, is the condition called "back-knee." This results from an abnormal amount of force being transmitted through the anterior segment of the growing lines in a position of rest.

In such positions of activity as are occupied in running, you see that the plumb-line falls very considerably internal to the knee-joint in a vertical

Fig. 13 represents the knee-joint as seen from one side, and from the position of the lateral ligaments, and of the transverse axis of rotation of the lower end of the femur, it is apparent that in the easy erect posture, the anterior portion of the growing lines transmits much more pressure than the posterior part, and when this posture is habitually assumed, the conditions figured in 14 result.

13   14

transverse plane, during which period the several portions of the growing lines are exposed to conditions of pressure, the reverse of those existing in the resting posture.

Therefore, to remove this deformity by other means than operation, it is necessary to apply a mechanical contrivance which shall act in such a manner that the amount of pressure transmitted through the several portions of the growing line be reversed, and the consequent rate of bone formation altered till the normal form is obtained, while at the same time attempts are made to improve the patient's general nutrition, to make him avoid such attitudes of rest as were originally responsible for the development of the deformity, and to assume as constantly as possible attitudes of activity, and especially such as are antagonistic in their action to this particular attitude of rest. These same principles apply with equal truth to the treatment

of the other resting deformities, and will therefore not be recapitulated.

Now we will pass on to the consideration of flat-foot. *The attitude of activity of the foot is one of considerable adduction, or, as it is sometimes called, inversion.* The arches are raised, and the inner margin of the foot forms with that of its fellow an angle which is open backwards. You can see this to perfection in a very fast runner, whose well-developed and highly-trained muscles retain his feet in a position of extreme activity. Watch such a man walk or run, and you see that he turns his toes inwards to a considerable extent, so that the slightly concave inner margins of his feet form an angle open backwards. That boots should be made to fit the foot in such an attitude is obvious, though shoemakers insist on rendering the mechanics of the foot insecure by forcing the great toe outwards instead of allowing it to come inwards as it should. The form of the boot is of vital importance not only in the treatment of flat-foot, but of all the resting deformities, and it should receive the closest attention and care of the surgeon.

The position of rest of the foot is that of abduction, in which the arches are depressed while the inner margin of the foot is rendered convex, and forms with that of its fellow an angle of considerable size open forwards. *This position becoming fixed, and later exaggerated, is called flat-foot, which is, therefore, simply the fixation of the foot in its position of rest.* Adduction and abduction of the foot take place in the subastragaloid joint. It is taught in anatomical works that adduction and abduction take place to a considerable extent in the ankle-joint when in a position of flexion, but this I showed in a paper in the *Journal of Anatomy and Physiology*, April, 1888, to be absolutely false.

Movements of adduction and abduction of the foot take place entirely in the subastragaloid joint. The movement is not simply a rotation of the foot around the long axis of the astragalus, but in adduction it is associated with an elevation of the height of the arches of the foot, its inner margin being rendered distinctly concave, while in abduction the arches are depressed and the inner margin of the foot is rendered distinctly convex. To explain the causation of this, I must call your attention to a strong ligament which had escaped the attention of anatomists till I described it in a paper in the Guy's Hospital Reports for 1886-

1887, page 252. It is continuous with the inferior calcaneo-scaphoid ligament with which its fibres run parallel. It is attached to the anterior portion of the sustentaculum tali, and from this its fibres run forwards, upwards and outwards, to be attached to the tuberosity and to the upper margin of the

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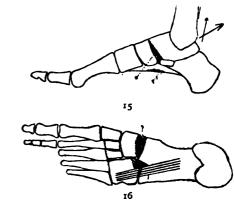
Fig. 15 represents the inner aspect of the foot, and fig. 16 its under surface, while in a position of adduction or of activity.

scaphoid. This I called the superior internal calcaneo-scaphoid ligament. You may remember that the tendon of the tibialis posticus, immediately before its insertion, sends a process downwards and backwards to the sustentaculum tali; when the tibialis posticus contracts, it, by this double insertion, besides tending to pull both bones upwards tends also to approximate them.

In drawing up the tubercle of the scaphoid, it winds up the superior internal and inferior calcaneo-scaphoid ligaments, approximating therefore the sustentaculum tali and scaphoid, and displacing the head of the astragalus outward upon the greater

Fig. 17 represents on the upper surface of the greater process of the os calcis, the facet B, which results from the habitual assumption of the active posture of the foot, associated with the transmission through it of a considerable pressure.

process of the os calcis, where, in labourers who are in the habit of carrying heavy loads, a new joint is developed, for obvious economical purposes.

The rotation of the os calcis results in a partial forcible separation of the opposing concavo-convex surfaces of the os calcis and cuboid, which, being opposed by the long and short ligaments, results in an elevation of the outer arch also.

In abduction, the sustentaculum tali and tuberosity being free from the strain exerted by the tendon of the tibialis posticus, fall apart, partly on account of the cessation of the traction

exerted by the bifid tendon and partly because the consequent rotation of the scaphoid upon the astragalus is followed by an unwinding of the calcaneo-scaphoid capsule, resulting in its *ap-*

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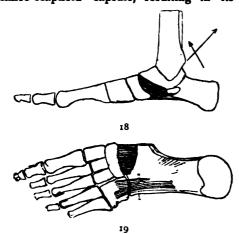
Fig. 18 represents the foot from the inner aspect, and from the same in the posture of rest or of abduction.

parent lengthening. This apparent lengthening has suggested the explanation of flat-foot which is usually given, namely, that it is due to yielding of the inferior calcaneo-scaphoid ligament which is described as being an elastic structure. This is obviously absurd. The head of the astragalus occupies the increased interval between the tuberosity of the scaphoid and the sustentaculum tali, and the opposing surfaces of the cuboid and os calcis coming into accurate apposition, the outer arch reaches its limit of depression.

When the foot is retained habitually in the attitude of rest, or of abduction, it becomes fixed in that position, the patient experiencing pain in that portion of the head of the astragalus which is only saved from direct pressure by the unwound calcaneo-scaphoid capsule, and it is pain of the same character as that felt in hallux valgus, owing to abnormal pressure being exerted upon the articular surface of the inner portion of the head of the bone. Pain is also felt in the upper part of the calcaneo-cuboid joint owing to stress of opposing bony margins, and also externally owing to the impaction of the external malleolus upon the os calcis. The fixation of the foot is, in early cases, due to the violent contraction of the abductor muscles which oppose the movement of adduction because it is painful, and in later cases adduction is opposed by changes in the form of the bones and joints of the nature of a mechanical arthritis.

The *treatment* of most cases is obvious. Under

an anæsthetic force the foot into an extreme position of adduction or of activity, and retain it for a considerable period immovably in it. Later, educate the patient to walk on the foot in the same position, namely, one of varus. If it is possible to continue this treatment under favourable circumstances, the patient will regain the normal physiological functions of his foot.

In some cases the resting posture results from some painful affection of the part, as, for instance, rheumatism. When a portion of the body is affected with rheumatism it is at once placed in a position of rest.

Again, if an injury of the part is sustained the patient obtains much greater relief when the limb is in the position of perfect rest, consequently, it is assumed; and as the rest is often necessarily prolonged, fixation of the foot in that position results.

These and similar causes for the prolonged assumption of the position of rest of the foot must, therefore, be taken into consideration in the treatment of so-called flat-foot.

In any case, obtain suitable boots with low heels and with the inner margin straight or slightly concave. The presence of a separate compartment for the great toe as used by one firm of bootmakers, is of additional service. To attempt to prop up what is called the arch of the foot permanently by pads, springs, etc., appears to be quite as unscientific as the use of jackets and other apparatus in lateral curvature.

We will pass on now to the consideration of dorsal excurvation and lateral curvation.

The term dorsal excurvation of young life, or the more popular one of *round shoulders,* are applied to a condition which results from the prolonged habitual assumption of the symmetrical posture of rest without the correction which a combination with the active erect attitude affords.

Strip the patient, and on examination you observe that the dorsal spine is *flexed to its extreme limit,* the head being displaced forwards and on a much lower plane than that occupied in the active erect posture, the clavicles slope forwards and downwards, and the scapula rests on the side of the chest rather than on its posterior aspect. By this means the angular prominence formed by the shoulder is reduced to a minimum and gives rise to the term "round shoulders."

Pass a tape measure around the thorax at the level of the nipples, and you will see that during

ordinary respiration the difference between the thoracic movements in expiration and inspiration are practically nil, that *the chest being in a position of almost complete expiration*, and that the breathing is being carried on almost entirely by means of the diaphragm. Though this is par-
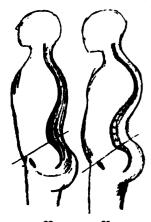
Fig. 20 represents a vertical antero - posterior section through the spine in a position of activity, namely, the active erect posture; and fig. 21 a similar section in a position of rest, which, when fixed by its habitual assumption, is called "dorsal ex-curvation of adult life" or "round shoulders."

20   21

ticularly marked in this condition and in lateral curvature, it exists to a less degree in patients showing any of the other resting deformities.

The convexity of the lumbar curve is diminished so that the whole length of the spinal column forms a long single curve, the summit of whose convexity corresponds to the mid-dorsal region, while the pelvis is over-extended upon the femora if the patient is standing.

If the skeleton of a typical case be examined, the bodies of the dorsal vertebræ are seen on vertical antero-posterior section to be elongated slightly from before backing, and to be wedge-

Fig. 22 represents in a vertical antero-posterior section through the summit of the dorsal curve the changes that take place in the shapes of the bodies of the vertebræ, and in the thickness and character of the fibro-cartilages in the condition called dorsal incurvation when of long duration.

shaped, the base of the wedge being directed backwards, its depth being in excess of the normal measurement of the posterior surface of the body, while the anterior surface of the body is very considerably under its normal depth. This fixation of the dorsal spine, together with the corresponding depression of the ribs and sternum, render

it impossible to raise the clavicles and scapula into the position which these bones should occupy when in the active erect posture. The conjugate of the brim of the pelvis is slightly diminished, owing to a yielding of the sacrum around a transverse axis passing through its centre, the promontory passing downwards and forwards. This alteration in the conjugate of the brim is of importance in the female subject. It is very trifling in amount in the non-rachitic subject, but should this bone be softened by imperfect nutrition, this condition may be a very important feature of the deformity. These conditions are all shown in a very exaggerated condition in feeble old age, and they are very well illustrated by figs. 23, 24, and 25.

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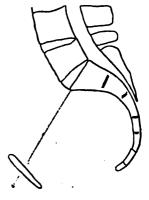
Fig. 23 shows the trunk of a feeble old subject in vertical antero-posterior section, and fig. 24 a transverse section through the thorax of the same case.

23

Fig. 25 represents a condition of the pelvis which at first sight closely simulates that associated with dorsal excurvation, namely, the diminution in the diameter of the conjugate of the brim of the pelvis, and the altered inclination of the facet on the first piece of the sacrum. This, however, is due to a separation or dissociation of the first piece of the sacrum to form one vertebra in excess above what are apparently the entire sacral series.

25

What happens as regards the spinal column during the assumption of this position of rest from the active erect posture is briefly a rotation of the

pelvis around a transverse axis in a direction the reverse of that undergone by each of the superjacent vertebræ with the changes resulting from deviations from the normal in the rate of bone formation at the several portions of epiphysial lines in consequence of the abnormal pressures to which they are exposed. When the bones have altered in form, but little can be done to remedy the deformity, but in cases of moderate severity teaching the patient to breathe deeply, by expanding the chest to its utmost, and then expelling forcibly as much air as possible during expiration, will not only open out the dorsal curve and increase the size and capacity of the chest, but it will oxygenate the blood and tissues as thoroughly as possible, and so improve the vigour and vitality of the individual. *In fact, it is impossible for the normal subject to retain the body in the positions of rest which become fixed as dorsal excurvation and lateral curvature where a very deep inspiratory effort is made.* This exercise, with a view of habitually changing as much air as possible during respiration, is of equally great importance in the treatment of all the resting deformities, but in the very young subject it may tax very much the patience and perseverance of the mother or nurse to enforce it.

Gymnastic exercises, the habitual assumption of the active erect posture, the avoidance of the particular position of rest which was primarily responsible for the deformity, plenty of real air, rest in the recumbent posture, tonics, sea-bathing, etc., will all be found of service in removing the deformity and in improving the patient's general condition and physique.

The detailed explanation of the physiology, anatomy, and causation of lateral curvature is, perhaps, more difficult to understand than that of the other resting deformities. I will state it as briefly as I possibly can, and if you wish to study the subject more fully I will refer you to a paper of mine on the subject in the *Transactions of the Medical Chirurgical Societies*, 1889, entitled "The Causation and Pathology of Lateral Curvation of the Spinal Column."

If the easy erect posture be assumed, the individual, standing on one leg, usually the right, the other knee being kept bent, the pelvis is rotated around two axes. It is over-extended upon the right femur, so moving around a transverse axis. At the same time it is fixed in a position of extreme adduction upon that bone, so moving around an

anterior posterior axis. The result of this double movement is that the plane of the sacral facet moves around an oblique axis intermediate between these, its surface being made to look backwards and to the left. Each of the superjacent vertebral discs rotates around a similar oblique axis, the degree of obliquity of the axis varying with the locality, since the presence of the ribs and the relation of the bony to the fibro-cartilaginous discs determine the formation, not of one single curve in an oblique plane, but of three curves in which are practically two planes. Since the axis around which each body moves is an oblique one, the apex of the spinous process does not deviate from the middle line to an extent which at all approaches the deviation of the centre of the body of each vertebra. Consequently the leverage action exerted by the transverse process and bodies of the vertebræ in the dorsal region produces the remarkable alteration in the form of the thorax with which we are so familiar.

A slight diminution of the conjugate of the brim of the pelvis is sometimes observed for the same reason as in dorsal excurvation.

Perhaps I may put this more simply. A rotation of the pelvis around an antero-posterior axis alone would produce a rotation of each of the superjacent bodies around an antero-posterior axis, and three curves would be developed in a plane at right angles to the axis of rotation.

A rotation of the pelvis round a transverse axis, such as takes place in over-extension of the spine on the thigh bones, produces of necessity a rotation of the superjacent bodies round a transverse axis. This results in the opening out of the lumbar curve with an increase of the dorsal curve.

In the rotation around an antero-posterior axis the segment of the fibro-cartilage corresponding to the concavity of the curve is more compressed than that on convexity.

In the rotation around a transverse axis the anterior half of the fibro-cartilage is more compressed than the posterior.

Consequently, in the concavity of lateral curve the segment of fibro-cartilage which is most compressed is the antero-lateral, while the postero-lateral, on the side of the convexity, sustains the least.

In rickets, whose most obvious characteristic is perhaps such imperfect nutrition of the bony system as results from indigestion in young life, *the epiphysial line, which is large, and irregular in*

outline, reacts to pressure in a much more marked manner than does the epiphysial line of the child which is merely wanting in vigour. Consequently when the bones are rachitic, the changes already described in the ends of the bones are much exaggerated.

But besides the changes in the lines owing to a general want of firmness of the diaphyses, these alter in form as well, and the deformity is so rendered more considerable. Besides those already described, there are certain deformities that are peculiar to the rickety child.

As the rickety child assumes the sedentary posture, the thorax is flexed abruptly upon the lumbar spine, and the lumbo-sacral and sacro-iliac joints are in a position of very considerable flexion.*
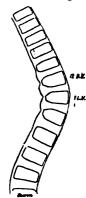
Fig. 27 represents a vertical antero-posterior section through the dorsi-lumbar spine from a case of rachitic spinal deformity. It shows the changes in the form of the lower dorsal and upper lumbar bodies, and the excessive proportion of fibro-cartilage.

The constant assumption of this attitude results in the eleventh and twelfth dorsal and the first and second lumbar vertebral bodies assuming a wedge shape, an angular curvature of more or less abruptness resulting. The sacrum yields around a transverse axis, and the posterior portions of the iliac bones are displaced forwards, producing a considerable diminution of the conjugate diameter of the brim of the true pelvis, and its peculiarly transversely oval form. This deformity is sometimes spoken of as rickety spine or kyphosis, or as dorsal excurvation of infancy. A similar, though very much 'slighter, degree of deformity may develop in the non-rachitic under the same mechanical influences.

When knock-knee develops in a rachitic subject the changes in the adjacent ends of the tibia and femur are associated with a yielding of the shafts of these bones, so that they together form a fairly uniform curve with its convexity directed inwards. If the development of the knock-knee be preceded by the deformity of the pelvis already

* See vol. ii., p. 183, of "The Clinical Journal."

described, there exists in the erect posture, owing to a hyper-extension of the hip-joints, a necessary flexion of the knee-joints. Under these circumstances the plane in which the curve formed by the femur and tibia lies runs obliquely inwards and forwards, and not directly inwards.

Bow-legs are developed only in the rickety subject, and at a much earlier period than the deformity just described. It depends upon a yielding of the softened diaphyses of the femur and tibia separately, and to some extent independently of one another, and in a vertical transverse plane during the period that the child is learning to stand. The epiphysial lines are not markedly modified in the rates of growth of their several segments till the child has acquired the habit of standing and walking. Then they react to abnormal pressure in the manner described in knock-knee, but in a reverse direction, the growth of the inner condyle being subnormal and that of the outer condyle excessive. The patella is displaced inwards till it may rest upon the inner surface of the internal condyle. If the development of this deformity be preceded by the forward displacement of the lumbo-sacral joint, the convexity formed by the femur and tibia is directed outwards and forwards, and not directly outwards.

A CLINICAL LECTURE

ON

NEURALGIC AFFECTIONS OF THE LIMBS.

Delivered at the West London Hospital by

W. ALDREN TURNER, M.D., M.R.C.P.,

Assistant-Physician to the Hospital; and Physician to Out-Patients, Hospital for Epilepsy and Paralysis.

GENTLEMEN,—The conditions to which I wish to direct your attention this afternoon are brachial neuritis and brachial neuralgia or *brachialgia*, and sciatic neuritis or *sciatica*. It may appear to you that the terms "neuralgia" and "neuritis" are being here used synonymously. It seems preferable, however, to confine the term "neuralgia" to the symptom of pain in a nerve, and the term "neuritis" to the pathological condition of nerve-inflammation. Neuritis gives rise, therefore, to neuralgia; but all neuralgias are not due to neuritis.

Sciatica is, I believe, invariably due to inflam-

mation of the sciatic nerve or its sheath ; and brachial neuralgia, at all events in its more severe forms, is undoubtedly a symptom of a neuritis of one or more branches of the brachial plexus.

The chief difference in physical signs between neuritis and neuralgia simplex lies in the presence of trophic disturbances, either of the skin, or more commonly, of the muscles, in the former condition. Trophic changes, the result of an apparent neuralgia, most probably indicate some pathological change in the nerve trunk.

A symptom common to both troubles is the presence of certain tender points in the lines of position of the nerves of the limbs. Nerves are most liable to tenderness on pressure, where they issue from deeper parts through either bony canals or fibrous rings. We are therefore able to locate with accuracy the spots where such tenderness should occur. These points are, in the *upper limbs*, at the places where the following branches pierce the deep fascia :—

The branches of the circumflex nerve over the deltoid muscle.

The external cutaneous branch of the musculo-spiral, in the middle of the upper arm posteriorly.

The external cutaneous branch of the musculo-cutaneous over the supinator longus muscle.

The palmar cutaneous branch of the median ; and

The dorsal cutaneous branch of the ulnar nerve at the front and back of the wrist respectively.

In the *lower limbs* :—

The gluteal branches of the sacral plexus over the gluteus maximus muscle.

The sciatic branches on the posterior aspect of the thigh.

The peroneal nerve below the head of the fibula.

The malleolar branch below the external ankle; and

The external plantar branch on the outer side of the foot.

In the neuritic cases there is marked and severe pain on pressure along the trunks of the nerves in addition to the afore-mentioned tender points.

The tenderness of the nerve-trunks is readily explained by the presence of an inflammatory condition involving the nerve and its branches ; but the explanation of the tender points, especially in cases where the idea of a neuritis is not entertainable, presents greater difficulties. It is not improbable that, in many of these cases, the irritation is caused by the presence of an inflammatory or other change in the fibrous tissue through which the nerve passes. It is well known, for example, that the most intense occipital neuralgia may be caused by the presence of a small fibrous tumour or localised fibrous thickening at the place, where the great occipital nerve pierces the deep fascia of the neck.

It is not uninteresting to look for one moment at the relative frequency with which neuralgic affections occur. Of 23 cases of neuralgic affection of the limbs, of which I have notes, 16 are cases of the cervico-brachial form, the remaining 7 including crural neuralgia, sciatica, and lumbago.

Of the 16 brachial cases, 4 are cases of neuritis, 3 of which occurred in men. Of the remaining arm cases, the remainder were in women at or about middle life, with one exception, which was in a male patient, who also happened to suffer from epileptic vertigo.

A well-marked case of severe *brachial neuritis* presents a fairly typical picture. The patient complains of intense pain in the arm, chiefly about the shoulder-joint, and much aggravated by movement. Complaint, also, is often made of a subscapular pain, not only in the idiopathic form under consideration, but also in those which occur after a fall on the shoulder. This pain is more or less constant with paroxysmal exacerbations. These are especially severe and troublesome at night so that sleep becomes impossible. A physical examination shows that passive movement of the arm is productive of as severe pain as is voluntary movement ; pressure along the nerves also gives rise to much suffering, and the points, which have been already indicated are, on pressure, the seat of intense pain. The muscles are usually soft and flabby, and may be distinctly atrophied in groups, such as, for example, the thenar and hypothenar eminences.

A man, aged 54, farmer by occupation, complained of difficulty in moving the left arm. He stated that movement gave rise to severe pain in the neck, shoulder and arm on the left side ; slight and feeble movements could, however, be effected without much pain, which was usually constant.

Severe pain was elicited on pressure over the brachial plexus, above the clavicle and in the axilla, as well as along the nerve trunks in the upper arm ; and there were points tender on pressure over the deltoid, external cutaneous, median, musculo-spiral and ulnar branches.

There was some wasting of the small muscles of the hand, chiefly of the thenar and hypothenar

eminences, and the patient wore a woollen glove upon his left hand, which, he found, gave a certain amount of relief. The circulatory and urinary systems were normal, and he never had gout; on the other hand, he suffered much from lumbago and rheumatic pains, and he was exposed to all sorts of weather.

He had suffered from this condition for nearly two years, and had tried all the known remedies, as well as the waters of Buxton, without much relief.

Although this condition would appear to present no special diagnostic difficulty, yet it is one very easily and very commonly mistaken. If it has existed for some length of time, there arise inflammatory conditions in the shoulder-joint leading to adhesions and secondary atrophy of the muscles; in this way an appearance of disease of the joint with "arthritic" muscular atrophy may be set up, and the original condition rendered obscure. An analogous condition is also met with in cases of old-standing hemiplegia, where the slightest attempt at movement gives rise to severe pain in the arm and shoulder; this form has also well-marked tender points in the usual situations.

Of four well-marked instances of brachial neuritis which I have seen, one gave a distinct history of acute gout, while the others, although subject to rheumatic pains and pains in the back, did not give any history of acute rheumatism or gout. There seems to be little doubt, however, that this form of neuritis is especially liable to occur in those who are the subjects of gout, just as a minor variety, e.g., localised neuritis of the ulnar nerve, occurs most commonly in persons thus disposed; the fact also that brachial neuritis is commoner in men than in women may be similarly explained.

But it is the milder forms of the affection to which I wish more especially to direct your attention. In many such cases we have to deal with a purely neuralgic affection, there being no extensive inflammatory condition of the nerve sheath. The condition is, I believe, very common, often overlooked and readily misunderstood. The patients, who are usually women of middle age, complain of "rheumatic pains" in the arms. I have even known a patient state that she has lost the power of the arm, thereby indicating that owing to the pain on movement the arm was not of much use to her. In all cases of so-called "rheumatics," it is advisable to make a physical examination, for *pain* (the symptom) has, in the majority of in-

stances, some objective basis. It is not unlikely also, that as the condition under discussion is a troublesome one and tedious, the patient has previously sought advice elsewhere, and has probably been told to "drive away" the rheumatism by movement, gentle exercise, and so on; but has found that instead of driving it away, she has really driven it on.

On making an examination, it will be found that the movements of the arm are limited, owing to the pain which is caused thereby, this being chiefly seen on elevation of the arm. Gentle pressure will elicit pain in several areas, the commonest being, in my experience, over the external cutaneous branch of the musculo-cutaneous nerve. Other points are those already indicated, chiefly the deltoid branches of the circumflex, and the palmar cutaneous branch of the median.

Although met with in this simple condition, one not uncommonly finds that brachial neuralgia is merely a local element in a general condition of nervous asthenia. Hence the physician's attention is often in the first instance directed by the patient to pain in the head, either on the vertex or limited to the occipital region. Physical examination will here elicit the fact that the occipital nerves are tender on pressure, where they penetrate the cervical fascia. Pain also about the cervical plexus is not uncommon. This is characterised chiefly by tenderness to pressure along the posterior border of the sterno-mastoid muscle, with a highly characteristic extension of tenderness over the clavicle on to the skin of the breast. There may also be pain along the spine, and in some cases the pain extends into the legs.

These symptoms, then, indicate a condition of general neuralgia, of which the brachial form is only a localised element.

But the question may not unnaturally be asked, How is a *differential diagnosis* to be made between neuralgia, the result of a neuritis, and the simple form? (1) In brachial neuritis the pain is limited to the affected arm and shoulder; in simple neuralgia it is much more commonly bilateral, or, as already indicated, it may be merely a local element in a general condition. (2) Brachial neuritis is a rarer affection, and is most commonly met with in men; neuralgia is common, and met with in women. (3) In neuritis there is marked pain on pressure along the nerve trunks as well as over the tender points; in neuralgia, the pain is usually limited to the latter. Pain on movement

is a feature of both forms. (4) Neuritis is characterized by trophic alterations in. the skin or muscles, giving sometimes in the latter the reaction of degeneration. Neuralgia rarely, if ever, occasions such trophic conditions.

Before considering briefly the neuralgic affections of the lower limbs, I shall mention what I believe to be the best means of *treating* the upper limb cases, as they are essentially curable. When the patient first comes under observation, it is necessary to enjoin absolute and complete rest for the affected arm. In one sense it is fortunate that the condition is painful, for by this means that rest is obtained which is difficult to enforce in less painful states. The arm should be placed in a sling or bandaged to the side of the chest, so that movement, however slight, may be prevented. I do not consider it advisable to splint the arm, for the patient often finds that a slight change of position gives temporary relief, which cannot be otherwise obtained. So difficult is it at first to get a person to appreciate the value of rest that the cure is often indefinitely delayed.

The second great factor in the treatment of these cases is counter-irritation. This may be employed either in its mild or severe forms, beginning with mustard-leaves and passing up to the Pacquelin thermo-cautery. It is best to apply the counter-irritant over the tender points. As one application is not usually sufficient, it may be necessary to repeat the local application on several occasions.

The third factor in the treatment is the administration of internal remedies to counteract the diathetic tendency. I have obtained the most satisfactory results from a combination of salicylate of soda with iodide and bromide of potash; while the purely neuralgic cases are best treated with arsenic, iron, and quinine.

In discussing the *neuralgic affections of the lower limbs*, we have to deal with conditions analogous to those already met with in the arms. There is sciatic neuritis, giving rise to sciatica, a true inflammatory condition of the sciatic nerve and its sheath. There are, in addition, several forms of neuralgia simplex, occurring in the nervously asthenic person, the commonest of which is crural neuralgia, although these seem to be less common than in the upper limbs.

In acute sciatica the slightest movement gives rise to severe pain. The leg is maintained in a characteristically stiff and flexed position. There

is marked pain on pressure along the sciatic nerve and over the points already mentioned. These are usually the sciatic notch, the middle of the thigh posteriorly, below the head of the fibula, and below the external malleolus. The affected limb may become distinctly smaller from muscular wasting, and there may even be an alteration in electrical excitability, rarely, however, amounting to a qualitative change such as the "reaction of degeneration."

The chief difficulty in connection with pain in the sacro-iliac and thigh regions lies in the question of differential diagnosis. In all such cases a physical examination of the back, and usually of the rectum, should be made.

The conditions to be specially borne in mind are :—

(1) Disease of the hip-joint.

(2) Neuralgic affections of the gluteal nerves, and those about the iliac crest. These present the usual features of pain on movement and pain on pressure over certain points, which are over the lower border of the gluteus maximus, at the middle of the iliac crest, and in the scrotum.

(3) Disease of the sacro-iliac joint, a condition which is apt to be overlooked; and which, from the nature and distribution of the pain, may, in the early stages, readily be mistaken for neuralgia. The chief points of difference are, in disease of the joint, pain on pressure over the sacro-iliac synchondrosis, often extending into the groin, and pain on pressing the innominate bones together or on pulling them asunder. In the later stages, the swelling over the joint and bogginess on pressure, usually permit of an accurate diagnosis; moreover, this condition is unilateral. In neuralgia, on the other hand, the pain is not uncommonly bilateral, and there is no pain on lateral pressure; and, as I have endeavoured to show throughout, such neuralgia is often merely a localised element in a state of nervous asthenia; so that in such cases one would expect to find evidence of general nervous debility and neuralgia elsewhere.

The treatment should be conducted on the lines already laid down for similar affections of the arms. In addition to rest and counter-irritation, any diathetic tendency should be counteracted by suitable drugs. For the severe pain of lumbago and sciatica, hypodermic injections of morphia are most valuable; while relief may also be obtained by injection of chloroform, cocaine, and antipyrin into the muscle or sheath of the nerve.

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON TWO CASES OF

SYPHILITIC DISEASE OF THE SPINAL CORD.

Delivered on January 26, 1895,

By W. HALE-WHITE, M.D.,

Physician to Guy's Hospital.

GENTLEMEN,—We have, in John Ward, two excellent cases of spinal syphilis, which I think we may profitably study, because I have often demonstrated to you the chief symptoms they show.

The first case is that of a man, aged 57, who was admitted oh January 1st, 1895, for paralysis of both lower extremities. He had syphilis when a young man, but with this exception his health has been good till three weeks ago, when, as he was returning from the railway station, his legs seemed to give way under him. He managed to get home and went to bed, where he has remained ever since.

On admission. There are pigmented syphilitic scars on both arms and legs. All movements of the lower extremities are weak, and he cannot stand. There is no inco-ordination, and no variety of sensation is impaired. The muscles are flabby but not wasted, and do not show any reaction of degeneration. The knee-jerks are exaggerated, ankle clonus is present on both sides, and patella clonus on the left. There is some incontinence of both urine and fæces; except for these symptoms he is entirely healthy. He was treated with iodide of potassium, and is now taking 30 grains a day. Every day he has ½ grain of per-chloride of mercury injected into the gluteal region; he is kept in bed.

Now you notice that in this patient both legs are equally affected. That strongly suggests that the spinal cord is damaged, for were the lesion in the brain we should expect unilateral paralysis, and if in the nerves or muscles we should not expect the two legs to be equally affected. The supposition that it is not the nerves that are

diseased is at once confirmed by the fact that there is no pain nor tenderness in the lower ex-tremities. In every lesion of the spinal cord there are two things which you have to make out: one, what is the horizontal extent of the lesion? the other, what is its vertical extent?

Take, first, the horizontal extent of the lesion, and in this diagram of a transverse section of the cord you will see that if sensations of touch and muscle sense cannot be conducted upwards the lesion is in the posterior columns, and if those of pain and temperature fail the antero-lateral as-cending tracts are implicated. If there are signs of descending degeneration in the lateral columns, they must be implicated by the lesion. If there are trophic changes the anterior cornua are affected. This man has not now any sensory symptoms, so his lesion does not at present affect the posterior part of the cord, if it did slightly in the first instance; but he has evidence of lateral sclerosis, so the lateral columns are implicated by it. As to the anterior cornua, you will re-member that he has incontinence of urine and fæces, that is to say, as fast as they pass into the bladder and rectum they pass on and out, because their presence there does not reflexly excite the sphincter vesicæ and the sphincter ani to act; or, in other words, the bladder and rectal reflexes are destroyed. We have seen that the posterior part of the cord is unaffected, consequently the anterior part which enters into these reflexes must be diseased, and so we know that the anterior cornua are damaged. Horizontally, therefore, there is a lesion extending over the anterior two-thirds of the spinal cord.

You learn the vertical extent of the lesion by observing the reflexes; for example, suppose in any case the cremasteric reflex to be absent, but the plantar and epigastric to be present, it is clear that the cord is diseased over an area including the cremasteric reflex centres, but not reaching down to the plantar reflex centres or up to the epigastric. In this case, as the vesical and rectal reflexes are destroyed, the lesion must be in the lumbar region of the cord. Also, the upward limits of the symptoms help us; for in-stance, you know that if anæsthesia extends no

higher than that part of the skin corresponding to the seventh dorsal segment, that the lesion does not extend upwards beyond that segment. Applying these principles to this case we conclude that the patient has a patch of myelitis in the lumbar cord of very small vertical extent, not implicating at the present time the posterior or antero-lateral columns. But inasmuch as both lower extremities are in a condition of spastic rigidity, and as the only evidence that the front of the cord is affected is the incontinence, we must suppose that the lesion has a greater vertical extent in its posterior than in its anterior part.

The next thing we have to consider is what is the lesion. You remember the symptoms came on suddenly. Now sudden onset means a vascular lesion. Apart from injury, it is excessively rare to find hæmorrhage into the cord unless it be part of an acute inflammation, or, in other words, a myelitis, and we are aware that myelitis is more common in those who have had syphilis than those who have not. Consequently, the most probable diagnosis in this man is that he has had an acute myelitis of slight extent, so slight, indeed, that it has not caused any pyrexia, and has damaged but a very small part of the cord. But you must also remember that just as the cerebral vessels are often affected with syphilitic arteritis and consequent thrombosis, so, perhaps, the spinal may be, and this man's lesion may be the result of such an arteritis.

Mild cases of myelitis like this, with paraplegia for the chief symptom, are by no means uncommon, and, especially in syphilitic subjects, the sensory symptoms may be very inconspicuous, and they usually clear up long before the power in the legs returns. Then, also, in syphilitic cases, as in this case, the patient often states that the onset is sudden.

I should not have mentioned trophic changes, for they are very rare, had it not been that you have recently seen, in Stephen, a man who came in for pyrexia with pain and distension of the left knee. He had rheumatic fever before and had an apical murmur, and the diagnosis had been made that he had rheumatic fever. You will remember that we withheld a diagnosis mainly because one joint only was implicated, and it was a good thing that we did, because salicylate of sodium did not relieve him, and the arthritis turned out to be trophic; he developed an acute trophic bedsore, incontinence of urine, paresis of legs, impairment

of some forms of sensation, and trophic arthritis of the ankles.

The exaggerated knee-jerk and ankle clonus you see in the patient before you are not difficult to interpret. Ankle clonus alone may occur in healthy people when they are tired or excited, or, in other words, when the impulses which usually proceed from the cerebrum and inhibit reflex processes are in abeyance. The knee-jerk may, as I have often pointed out, be exaggerated in the early stages of peripheral neuritis, but the two symptoms taken together always mean that there is sclerosis of the lateral columns. Why this is so it is difficult to explain; on one view there are constantly passing down the lateral tracts from the cerebrum, perhaps the optic thalamus, inhibitory impulses which restrain the activity of these reflexes, therefore, when owing to the lateral sclerosis they can no longer pass down the diseased columns, the reflexes are very lively. Others have supposed that the sclerosis of the minute terminal filaments of the fibres of the lateral column irritates the minute branches of the anterior cornual cells, and so increases their reflex excitability. It would take us far too long to go thoroughly into the vexed question of the mechanism of ankle clonus and the knee-jerk, nor is it necessary here, for clinically remember, that when ankle clonus and exaggerated knee-jerk occur together, there is usually, as here, spastic rigidity of the limbs, and when these symptoms are present you can always be sure that the lateral columns are sclerosed.

The second case is that of a man, aged 40, who was admitted December 22nd, 1894. Seven years ago he contracted syphilis, otherwise his personal history is unimportant. The present illness began at Christmas, 1893, with a pain in the back about the second and third dorsal spines. Gradually he became weak in the legs, and the dorsal pain became more severe, and spread round the chest. At the end of August he had retention of urine, and was unable to stand. He went into the hospital at Canterbury. His urine was drawn off for six weeks; then he had some incontinence, but now he has fair control over the bladder. Whilst in the Canterbury hospital he suffered much from constipation; he was given purgatives, but when the bowels were open he had no control over the rectum, and the fæces were passed involuntarily and unconsciously. Now he has moderately good control. Shortly after his admission at Canterbury

he had a constricting sensation round his chest, as though a rope were tied round it, and a bedsore appeared. At first his legs wasted much, but now they have almost regained their natural size.

Condition when admitted. The power of his lower extremities is satisfactory. There is no inco-ordination, and there are no abnormal sensory phenomena in the limbs, which are in a typical condition of spastic rigidity. The knee-jerks are very exaggerated, and ankle clonus is difficult to obtain on account of the extreme rigidity of the legs. The left arm is affected with old infantile paralysis. The right is weak, and the muscles of the shoulder are wasted. He complains of pain about the upper dorsal vertebræ, and just above the right nipple is a band two inches wide, over which there is loss of all varieties of sensation. This band begins at the sternum, but at the anterior border of the axilla, its upper and lower borders approach so that it ceases in the middle of the axilla, but there is continued round to the spine a narrow area, over which he says sensations are queer, and there is some numbness. The right half of the diaphragm and the right serratus magnus are almost completely paralysed; there is no scapular reflex. The pupils are equal. He was given a drachm and a half of liquor hydrargyri perchloridi and iodide of potassium in increasing doses.

On January 1st he was taking 30 grains thrice a day of the iodide, and by January 10th he was taking 45 grains thrice a day. On January 12th he showed some signs of iodism—viz., a typical rash, slight headache, and coryza. The dose of iodide was accordingly doubled by giving the same dose six times a day, or 370 grains a day. In forty-eight hours the rash had almost faded, and the other symptoms of iodism had disappeared.

The symptoms for which he was admitted have improved. He has now good control over his bladder; the right upper, the right serratus magnus, and the right half of the diaphragm are stronger, the pain is much less, and the anæsthetic area is not so distinct.

In this case there is a group of symptoms to which I now draw your attention, viz. :—

1. Pain in between the scapulæ.

2. Painful sense of constriction around the right side of the chest, above the nipple.

3. Zone of anæsthesia over most of the same area.

4. Absent scapular reflex.

5. Wasting of muscles of the right shoulder.

6. Weakness of the right arm.

7. Some paralysis of the right half of diaphragm.

8. Some paralysis of the right serratus magnus.

In the first place, you will notice that it is in the highest degree improbable that these symptoms can be due to any disease within the spinal cord, because they are unilateral. Any lesion in so small a structure as the cord could hardly be exclusively confined to one side; and further, the presence of pain shows that the lesion must be outside the cord, for lesions of the cord itself are not accompanied by pain. The lesion cannot be in the muscles, for not only are primary diseases of muscles very rare, but they are painless. Therefore there must be some interference with the functions of the nerves themselves. Next notice that the phrenic and the nerve to the serratus magnus, and to a less extent, as shown by the weakness and wasting of the upper extremity, and the loss of scapular reflex, the whole brachial plexus, and also the cutaneous branches of the nerves supplying the band of skin above the nipple, are implicated, and you see that the trunks of the lower cervical and upper dorsal nerves are affected before the phrenic and long thoracic nerves are given off, but these branch off almost directly the fourth, fifth, and sixth nerves leave the intervertebral foramina, and therefore the lesion must implicate the nerves very close to the cord. We know from the pain and paralysis that the lesion presses on both the posterior and anterior nerve roots, and the anatomical distribution of the symptoms tells us that those from the fourth cervical to about the third or fourth dorsal are implicated.

There are one or two interesting points to make a note of in passing; one is that the pressure is clearly unequal on different nerve roots, for there are no sensory symptoms down the arms; in fact, it looks as though the anterior roots were pressed upon higher up and the posterior lower down. Then you will observe that the pupillary fibres, leaving the cord by the first thoracic and ascending in the cervical sympathetic, are unaffected. This case, too, illustrates a well-known clinical fact, viz., that when chronic lesions press on nerve roots the evidence of irritation of the anterior nerve roots, that is to say, spasm of the various muscles, is often absent, and we get only signs of their compression and loss of function, that is to say, paresis of the muscles supplied by them. In the case of the posterior nerve roots it is different;

they more readily show evidence of irritation—that is, pain—along the course of their cutaneous filaments; but as pressure continues, and the posterior nerve root becomes incapable of conducting sensations, the pain is replaced by anæsthesia. Actual tenderness over the back is not uncommon; the most probable explanation of it is that the pressure of the hand is transmitted to the diseased nerve roots, although there is, of course, the possibility that it may have something to do with one of Head's tender areas, which I have lately had such excellent opportunities of demonstrating in the girl in 31 bed, Miriam. Occasionally a lesion of nerve roots produces cutaneous eruptions, but this is decidedly rare.

Lastly, you must remember that you must on no account conclude that because there is a band of anæsthesia only two inches wide that only one posterior dorsal root is affected; for we know that the sensory distribution of contiguous dorsal nerves overlaps so that each two inches of skin all over the body receives fibres from two or three dorsal nerves. Hence in this case we know that two or three dorsal posterior nerve roots are implicated. Before leaving these symptoms I should guard you against laying too much stress on the loss of the scapular reflex. I have mentioned it for uniformity, but it is often absent in health.

The next question is, What is the lesion of which we have just learnt the position? Well, as it is pressing on the nerve roots, it is probably in the meninges. We know from experience that chronic inflammation of the dura mater is nearly always due to disease of the vertebræ. As there is no evidence of this, we conclude that the pia-arachnoid is affected. You will observe that not only is this chronic inflammation of the pia-arachnoid very irregularly distributed here on the anterior nerve roots, there on the posterior, but that although there is considerable patch on one side of the cord there is, as far as our evidence goes, none on the other. This irregular distribution is very suggestive of syphilis, and as the man has had syphilis, it is almost certain that he is suffering from a syphilitic chronic spinal meningitis of the pia-arachnoid on the right side, from the fourth or fifth cervical nerves to the third or fourth dorsal. That means there is much chronic inflammatory or even gummatous thickening of the membranes; the mass of new deposit may in places be $\frac{1}{4}$ inch thick in some cases.

You have already learnt, from the first case, that

the presence of exaggerated knee-jerks, spastic rigidity and ankle clonus means that the patient has chronic descending sclerosis of the fibres of his pyramidal tracts, because their continuity is interrupted, and, consequently, the parts of them below the solution of continuity are cut off from their trophic cells in the motor cortex. There are two possible ways in which the chronic syphilitic meningitis might effect this, either the thickening of the pia-arachnoid might compress the cord, or the gummatous material might grow into the cord from the meninges and form a so-called gumma of the cord, but remember no such thing as a gumma of the cord or brain, in strict language, exists; what always happens is that the gummatous deposit extends inwards from the meninges. Whether the cord is compressed or whether the gummatous growth has extended in is very difficult to say. No doubt it surprises you that the latent tracts should have their continuity interfered with, while, at the same time, the posterior columns and the antero-lateral columns should be left free, for there is no sort of impairment of condition upwards of any sensory impressions from the legs. I admit it is surprising; I can only say that it is in accord with clinical experience, for it is no uncommon thing to meet with descending sclerosis in such a case as this without any impairment in upward conduction. It may be because, as already pointed out, in the spinal cord as in the brain, syphilitic thickenings of meninges are often very irregularly distributed, but you must remember that it may be because, when the usual path of sensations in the cord is blocked, they, without much difficulty, open up new paths for themselves in other parts of the cord, and this may explain why, in the first case, although the man had myelitis he had no sensory symptoms.

But there is another possibility to bear in mind, which would also explain the lateral sclerosis—that the patient had lower in the cord a small patch of myelitis, for we have seen that syphilitic subjects are very liable to myelitis. This would explain the wasting and loss of power which affected the legs five or six months ago, and, if the vesical and rectal centres in the lumbar region were also affected, the incontinence of urine and fæces which is said to have been present before he came to us would be explained; for, as the first case taught us, when myelitis so implicates these centres as to destroy their functions permanently or temporarily, owing to the failure of the reflex, the presence

of urine in the bladder or fæces in the rectum does not reflexly lead to contraction of the sphincter vesicæ or sphincter ani, and so the urine and fæces are discharged as fast as they pour into the bladder or rectum. Still it is not necessary on this account alone to suppose the presence of myelitis of the vesical and rectal centres, for sometimes when some damage to the cord high up prevents impulses from reaching the lumbar cord, if the sphincters have been used to much help from voluntary control there may be incontinence. But the fact that the man says he was sometimes unconscious of any action of the bladder or bowels, appears to show that there was some impairment upwards of sensory impressions, which might very well have been due to some myelitis. So that we may say that the wasting and loss of power of the lower extremities, incontinence and loss of sensation of the passage of urine and fæces, are strongly suggestive that earlier in the attack he had some syphilitic myelitis, which may be the cause of his spastic paraplegia; and we, remembering that syphilitic meningitis is often very patchy, must not lose sight of the fact that possibly these symptoms may have been due to extension from the meninges of a patch of meningitis lower down—an unlikely view, however, in the face of the fact that he never had any pain in the legs.

These two cases illustrate most admirably the effects of syphilis on the spinal cord, and also they show the points of difference between a myelitis and meningitis. They are, in the main, that pain and unilateral symptoms mean a lesion outside the cord. Then, too, each of these cases is typical of a common class of case. The first class, of syphilitic myelitis, which is usually of the lower part of the cord, is almost always accompanied by paraplegia and by vesical and rectal symptoms, but in which sensory symptoms may be absent and which, if we may judge by the slight constitutional disturbance and by the limited area involved, is a mild myelitis. It is not a syphilitic myelitis in the sense that there is a gummatous deposit in the cord, but in the same sense that locomotor ataxy is syphilitic; that is to say, persons who have had syphilis are more liable to myelitis than those who have not.

The prognosis is of the very greatest moment, and the most important symptoms from this point of view are those relating to the bladder, for if retention of urine leads to cystitis and consequent suppurative nephritis, the prognosis becomes ex-

tremely grave, and probably the patient will die from exhaustion, pyæmic infection, or lardaceous disease, and remember retention may easily be due to carelessness on your part.. Bedsores are equally important from a prognostic point of view, and here, again, your carelessness or that of the nurse may easily lead to the patient's death; for if a bedsore is not detected early and carefully treated, death may occur from exhaustion, pyæmia, lardaceous disease, or suppurative meningitis of the cord from erosion of the bones of the lower part of the spine. The prognosis, too, depends much upon the degree of respiratory paralysis; for if the myelitis or meningitis is extensive in the cervical or dorsal regions, the phrenic and intercostal nerves are affected, the patient's respiratory movements are hampered, and he gets œdema of the lungs and bronchitis, which kill him.

Other points for you to bear in mind are that the prognosis is good in proportion to the rate of improvement; it is better if the sensory symptoms are few and quickly pass away, than if they are intense and persistant; other things being equal, the less the area of the cord involved the better, and the prognosis is better if the dorsal area is affected rather than the lumbar or cervical. The prognosis in our second case is far better than in the first, for there is some hope that we may, by iodide of potassium, absorb the gummatous infiltration of the meninges, but mercury and iodide of potassium have little, if any, better effect upon the myelitis which occurs in syphilitic subjects than they have upon that which occurs in those who have not had syphilis. Still, in the first case, sensory symptoms are absent, the lesion is very limited, and there are no bedsores or cystitis, so that, as cases of myelitis go, the prognosis is good, and we shall have to be guided entirely by the rate of progress.

Treatment.—The most important point is, if retention has occurred, to avoid cystitis by the use of a scrupulously clean catheter, or, if cystitis is already present, to cure it by frequently washing out the bladder with boracic acid. Avoid bedsores by careful nursing, and you will have to be especially watchful if there is incontinence of urine or fæces. You will then find it useful to have plenty of antiseptic absorbent cotton-wool, which must be frequently changed, under the buttocks and in between the thighs. A urinal may be used if it does not irritate the penis.

The patient must of course be in bed. Some

think that local applications, such as poultices or ice-bags to the spine, were useful in the early stages, but they are of no benefit when the patient has reached the stage of our two cases. In the second case there is, as the improvement has demonstrated, no doubt that iodide of potassium in large doses is the correct remedy; particularly notice the large doses the patient can bear, and also that on doubling the dose of iodide stopped the symptoms of iodism. Whether iodide has any influence on myelitis, even if the patient has had syphilis, is very doubtful, but I always give it because there is always the possibility of some local gummatous meningitis outside the cord.

Mercury is given to our first patient as a general absorbent, not because I believe syphilitic myelitis is more easily influenced than other varieties. Often, as in this case, when you want to bring the patient rapidly and certainly under its influence, it is well to give it subcutaneously, as this is much less trouble and much more cleanly than fumigation or inunction. A very good way is to dissolve $\frac{1}{6}$ grain of perchloride of mercury in five minims of water, and inject it deep into the gluteal region once a day. This usually suffices to keep a patient just on the brink of salivation. Many other preparations of mercury for subcutaneous injection have been used, but I have given many hundred injections of the perchloride without any ill results, and have found the patients very quickly influenced, so I do not care to change. The spreading of the myelitis, even the minute fraction of an inch, may, by destroying the vesical centre, ultimately kill the patient; and that is why, if the mercury is to do any good, it is well to get the patient under it quickly, and for this means subcutaneous injection is more certain than administration by the mouth.

Permanganate of Potassium as an Antidote to Opium Poisoning.—Dissolve 8 grains in 1 ounce of water, and administer a teaspoonful every ten minutes or quarter of an hour, increasing the intervals with the improvement in the patient. Sixteen grains have thus been given with recovery.
(*Ind. Med. Rec.*)

The hypodermic injection of $\frac{1}{10}$ of a grain of morphin combined with $\frac{1}{100}$ of atropin is the most efficient stimulant in cases suffering from nightly attacks of cardiac asthma.

A CLINICAL LECTURE
ON A CASE OF
TUBAL GESTATION:
With Remarks on Early Diagnosis.

Delivered at St. George's Hospital, Dec. 19, 1894, by
W. R. DAKIN, M.D., M.R.C.P.,
Obstetric Physician to the Hospital.

GENTLEMEN,—I shall take for my subject to-day a case of tubal gestation which is now in Burton Ward, and which most of you have seen.

The patient, E. S., aged 32, married ten years, was sent to the hospital complaining of pain and a feeling of stiffness in both hips when walking, the left side being the one more affected. She also complained of pain in the lower part of the abdomen on the left side, which had lasted eight weeks. At the time the pain commenced she had not menstruated for five weeks. After that interval she lost a little blood. She believed she was pregnant, but that "the loss was due to fright." Five years ago, immediately after the birth of her last child, she had an attack of pleurisy, otherwise she had had good health. That attack of pleurisy may have been puerperal fever of some kind or other. Her family history was good. She had had two children, the eldest nine years and the youngest five years old. There was nothing special about her menstruation, and she says that after her attack of pleurisy following her last confinement it has been still quite regular, so that, probably, there was not any serious disease of the appendages started then. Ever since marriage, too, she has practically had no pain, and was always regular. She last menstruated properly thirteen weeks prior to her admission into the hospital, but for about eight weeks she has been losing a little continuously. There was no flooding, and no solid substance had passed that she knew of. Micturition had been natural until eight weeks before admission, but since then she has had bearing-down pain in passing water. There was no trouble about the action of her bowels.

I examined the patient on admission on October 23rd. The abdomen was rather full. There was considerable tenderness in the left ovarian region, where there was a distinct mass to be felt rising to

an inch below a line joining the navel to the middle of the crest.

The right side of her abdomen appeared normal. That is a thing to be noticed, because, as a matter of fact, I found the tubal gestation to be in the right tube. There was, however, some muscular resistance over the right hypogastrium. On examination of the breasts there was some dried secretion found on the right nipple, there was some serum to be squeezed out, and there were ten or twelve large follicles on the areola. The left breast also had the same appearances, but there was no serum to be squeezed out. Per vaginam, the cervix was found to be low down, in the axis of the trunk, in the middle line, and quite fixed. The cervix admitted a finger to the internal os, and this was not patulous ; there was softening for about a quarter of an inch of the tip. The right side of the pelvis was encroached upon by a mass extending from the left side. The body of the uterus could not be definitely made out, but it formed part of the mass which mostly lay to the left side ; and the left side of the pelvis was filled by a fairly well-defined more or less globular swelling, very slightly moveable, extremely tender, about the size of a cocoa-nut, continuous with the body of the uterus, and extending into Douglas' pouch. No contractions were felt in the mass. Per rectum, the mass was found to be altogether in front of the rectum. A diagnosis of tubal gestation was made, and abdominal section was performed two days afterwards.

At the operation I found the right tube was distended and had given way. Surrounding the tube were many adhesions, which seemed to have confined all the hæmorrhage there had been in their meshes. Great difficulty was experienced in getting the tube free. These adhesions, no doubt, accounted for the fact of the woman not having shock or collapse, as patients usually do when rupture occurs in a gravid tube. It was found that the cyst must have given way at some previous period, as there was a quantity of old blood-clot adherent in Douglas' pouch. It was a case of tubal pregnancy of the right tube, and it was removed in the usual way by ligaturing the base of it. On the other side of the pelvis, you will remember, there was a large mass, which turned out afterwards to be, I believe, a perimetric abscess. That I opened a few days ago per vaginam, and that was probably connected with previous tubal disease which possibly caused

the extra-uterine gestation. That is the whole history of the case. .

Commentary.—The points to be considered in this case are as follows :—In the first place, What is the sort of woman in whom, from her history, extra-uterine gestation is a not unlikely accident ? Either a woman married for some time who has been sterile, or one who has had a child some years ago, and still being married and capable of bearing children has been sterile—in fact, after some considerable period of sterility. This patient had not quite a typical history of that kind, because she did not have a child until five years after she was married, and then it was a full-time one, and the pregnancy was normal. The next child she had was four years after that. She was not by any means a woman who had children rapidly, so that her having gone for five years was nothing remarkable. She then stopped menstruating in the ordinary way, and her breasts became active. There was nothing particularly abnormal about the pregnancy except that she began to have pain, which lasted eight weeks. She went one week over her period, and she began to have pain, though I think it was not pain which was marked to any extent. Then she began to lose a little blood continuously. She did not have any gushes of blood, as patients do in typical cases, and she passed no solid substance, or decidua, that she knows of—and she possibly did not ; it was simply a discharge of blood.

With regard to the pleurisy which she had after her last confinement, that may or may not have been septic and suggestive of concurrent tubal disease. She had at the operation considerable inflammatory disease in her pelvis, for on opening it we found a mass on the left side which formed part of the tumour noted at the vaginal examination. At the operation, however, it was not removed, as it appeared to be mainly composed of the body of the enlarged uterus, in the midst of thick adhesions.

Most cases of early tubal gestation are not diagnosed until after marked signs of rupture have appeared. We may say this case was diagnosed before extensive rupture had occurred, because no symptoms of internal hæmorrhage had been noticed. It is by the symptoms of hæmorrhage and shock that rupture is diagnosed.

Supposing we consider the typical history of a case of tubal gestation. The woman as a rule has menstruated regularly for, we will say, some years

previously, having been sterile to the degree I mentioned just now. She then misses a period and possibly thinks she is pregnant. After say six or seven weeks of pregnancy she begins to lose blood per vaginam in an irregular manner; she may do that on several occasions, and on one of these occasions she becomes collapsed out of all proportion to the vaginal hæmorrhage, and possibly, in a serious case, dies straight away in a few hours, if she does not have surgical relief; or the blood, which has been effused into her peritoneal cavity by rupture, or by escape of the ovum from the abdominal end of the tube as a mole, may coagulate and form one of the large intra-peritoneal hæmatoceles that are occasionally met with; or rupture may take place between the layers of the broad ligament, and be thus limited in amount. In the blood she passes per vaginam there is very often found a decidual cast, sometimes an entire cast of the uterus, or perhaps the decidua passes away in good large shreds. You have to distinguish that decidua from various other substances that may be passed by the vagina, but those I cannot at this time refer to.

There is one thing which you must not forget, and that is, that a decidua is thrown off in the case of abortion which may be indistinguishable from that of tubal gestation when shreds are examined microscopically.

After the ovum has become implanted in the tube, this is said to thicken for a few weeks during the growth of the ovum, and then to thin. It is always found to be more vascular, and large blood-vessels develop in it—large blood spaces are formed in its walls, and decidual cells are developed, so that there is more or less decidua formed in the tube. There are, however, no real decidual vascular spaces in the tube at all in which the villi as they grow can float, so that naturally a larger surface is necessary for the placenta than in intra-uterine pregnancy, if the ovum is allowed to grow for so long a period as to begin forming a placenta. The history of these cases, as understood now, is somewhat different from what it was supposed to be even five or six years ago. Then one had to describe many kinds of extra-uterine gestation, each of which was supposed to be able to arise primarily. First of all there was the case where the ovum lodged in the intra-mural part of the tube, and interstitial pregnancy resulted. This variety is established. Then there was tubal gestation proper —that is, gestation in the part of the tube lying

in the broad ligament. Then there were tubo-ovarian gestation, ovarian gestation, and primary abdominal gestation.

Let us contrast these with the present view of the subject. There is believed now to be really no such thing as primary abdominal gestation. Interstitial and ordinary tubal gestations are considered to be the origin of all the varieties of extra-uterine gestation. A tubal gestation grows, and when it reaches a period from about the eighth to the fourteenth week it ruptures; and it may rupture into the abdominal cavity or into the broad ligament. If it ruptures through the upper part of the tube into the abdominal cavity the blood is extravasated in that direction. The ovum escapes and dies in the middle of the hæmatocele which is formed there, if the woman still lives. Or instead of the ovum becoming entirely free, it may remain attached to the wall of the tube, and (as happened in this case) may be surrounded by blood clot, which is kept in position by adhesions or by insufficiency of opening in the tube; and then may go on developing for a little while longer, tearing through a little more now and then, as the adhesions which are formed stretch and give way, and finally probably ending in a large rupture, which is sufficiently distinctive, and may cost the woman her life.

If it ruptures the other way quite a different state of things arises. It ruptures down into the connective tissue of the broad ligament, of which the two layers are separated by the growing tube. Rupture here may be more or less gradual, just as it is in rupture through the free surface.

The result of the case now depends very much on the position of the incipient placenta. If this is on the upper part of the ovum, on the free aspect of the tube, then it is the embryo which is forced into the connective tissue of the broad ligament, and burrows into its substance, and goes on growing there for a certain time. Usually, after a certain variable period, if the ovum goes on growing, the broad ligament cannot expand any further, and ruptures. This is called secondary rupture, and is nearly always fatal. You will understand that it must be so because the placenta is at the top and forms a sort of roof to the whole mass, and any rupture must either go through it or dislodge it and rupture the large vessels which supply it with blood.

Supposing instead, that the placenta or the part that is going to form the placenta is the lowest

part of the ovum; it is, on rupture occurring, pressed down into the connective tissue of the broad ligament, the embryo remaining uppermost. The placenta can then make fresh connections in the intra-ligamentous tissue, and is thus enabled still to supply the embryo with blood. The embryo may go on growing now, and when it gets too large for the broad ligament to hold it, rupture occurs, and the fœtus is set free in the abdominal cavity, whereas the placenta remains attached to the portion of the tube which remains behind, and the connective tissue in the base of the broad ligament. In that case you have the fœtus free among the intestines, and the placenta attached to the broad ligament, nourishing the embryo in the ordinary way; this corresponds to the "abdominal" form. We need not go on further with what happens to the fœtus after it gets into the abdominal cavity.

I think I might now return to the physical signs of the mass. Notice its want of mobility; it was fixed like an inflammatory swelling. The cervix was slightly softened here, although it is seldom that you get softening of the cervix as early as this in extra-uterine gestation. There were no contractions to be felt in the tumour. Contractions are a most valuable sign, and should always be looked for in cases where the gravid uterus is possibly constituting the greater part of a tumour; you do not get contractions in an extra-uterine sac. There was a considerable amount of tenderness about it, showing that there was perimetritis present in a more or less active condition.
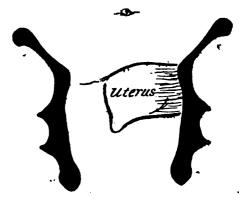
The diagnosis of these cases is the most essential point for you to consider; it is really the most important part of the subject, and you yourselves will have to make it, whoever does the operation afterwards.

All the cases we have had in the hospital have been sent up by medical men; and all those recently sent have been suspected or actually diagnosed; the patients have not come up of their own accord. You have to diagnose these cases, if you can, before rupture; at all events you must recognise the signs of rupture. In nearly all of them there are some signs of pregnancy; these may, however, be absent, and there may be only some hæmorrhage, and a mass which is growing; and there is, as a rule, some pain.

Take a case where the uterus is enlarged, and

in the second or third month of gestation. In diagnosing extra-uterine gestation it is as well to divide pregnancy into two periods: the first part up to the middle of the fourth month, where there may be a question whether pregnancy exists at all, either extra- or intra-uterine; and the second period, the last five months, when there is usually no difficulty about diagnosing pregnancy, and when you have to settle whether the pregnancy is in the uterus or outside.

Supposing, now, you have a case of normal uterine pregnancy between the second and third months; you have the uterus enlarged, it has not yet reached above the brim, and it is antiflexed. There should be no difficulty about diagnosing the pregnancy then, although in the second month, of course, there may often be some doubt. Instead, however, of having it central, free, and moveable, you find it over on one side and fixed. When a woman has suffered from perimetritis, her womb may be all over on one side, and for a time the
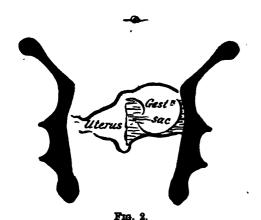
FIG. 1.

pregnancy will go on developing in that position (fig. 1). You may then have a tender mass which is more or less fixed, and the symptoms of early pregnancy.

There is not much difference in general shape between this and a tubal gestation. One point in such a case is to notice if, on bimanual examination, you get some contraction in the tumour. If you do, that settles the matter at once. You see in this case (fig. 2), where the tube only is enlarged, there could not possibly be any contraction in the mass. Another thing about extra-uterine gestation, as distinguishing it from intra-uterine, is that there are no hæmorrhages as a rule in the latter. The woman's breasts begin to

secrete in the ordinary way, and although there may be some pain there is no great amount of it. In *this* case, however, there is constantly increasing pain, and though menstruation often ceases, this sign is very irregular. The breasts often.have not begun to secrete, and the cervix is not soft, as I just now mentioned.

Fig. 2.

Another kind of case is where the uterus is pregnant between the second and third months, and is retroverted and fixed. There are signs of pregnancy, very likely some pelvic pain or sense of bearing-down, and some interference with the bladder functions may be beginning. The mass is immoveable, and lies in Douglas's pouch.

The diagnosis depends upon the same conditions as in the last case; the normal degree of softness of the cervix is present, and the pregnancy, for the present, may go on in the ordinary way. It may be possible to elicit contractions of the uterus. It may be impossible in this case to make a diagnosis.

Then, a third class is the case where the woman is in the act of aborting or has just aborted. In that case there is a possibility that it may be mistaken for extra-uterine gestation if the uterus be fixed by inflammation, acute and recent, or subacute. If the ovum has been saved there is no difficulty; but I am speaking of cases where only blood has been noticed by the patient. Possibly some shreds of membrane may be found. In cases of abortion the decidual shreds which the patient passes are not to be distinguished from the shreds passed by a patient who has extra-uterine gestation. But if chorionic villi are found in any shred that makes the diagnosis of abortion certain. She loses a fairly large quantity of blood, and on

that account she may be collapsed and very ill. The *history of the case* is what you have to go upon to a very large extent; the history of a patient normally pregnant up to the time of the miscarriage; the possible cause of the miscarriage, and the *shape of the uterus*. The uterus after it has emptied itself of an ovum is large and flattened. If you make out that the uterus itself is large and globular, large enough to constitute the greater part of the mass that you feel, your diagnosis will be in favour of abortion, threatened or proceeding. It is not always easy to at once distinguish between the two stages of abortion.

Then there are two other kinds of case that are important; one is a fibroid or fibroids of the uterus, which is fixed. In that case you may have sudden hæmorrhages. There is no loss of decidua, and the history of previous menorrhagia is some guide; there are no signs of pregnancy as a rule, though you will remember that sometimes there is serum in the breasts, and the cervix is softened in the case of fibroids.

Then in addition to the fibroids there are also perimetritic and parametritic masses.

In these you have a fixed, tender mass, and there is often some uterine hæmorrhage in the early stage of inflammation, or there may be amenorrhœa. There are, however, no signs of pregnancy; there may be a cause found for the inflammation. The pain ceases soon if the patient is put to bed. There is no decidual discharge.

You see, therefore, that any case or phase of early pregnancy in which the uterus is fixed, *may* be mistaken for ectopic gestation; and it may, after even the most careful consideration, and after repeated examinations, be impossible to come to a confident diagnosis.

In some of the cases which I have mentioned you may pass a sound, and thus make out whether the uterus is enlarged or not; but that is a thing you should never do unless as a last resort, because the case may be one of intra-uterine gestation, only temporarily abnormal. In this case a sound was not passed, we relied entirely upon the history and physical signs, and we found that our diagnosis was correct.

In diagnosing cases of *tubal rupture* you have to distinguish them from all sorts of things which cause collapse and pain in the abdomen. Rupture of abdominal aneurysm or twisting of the gut in any way—twisting of the pedicle of an ovarian

cyst, which is a very possible mistake, or twisting of the pedicle of a broad ligament cyst, or of a hydrosalpinx, may be confused with rupture. In such cases you have to take the whole history into consideration. Then you may mistake a case of poisoning for rupture. One or two cases in the early part of this century, and more recently too, have been put down to poisoning, which upon examination have turned out to be cases of rupture of extra-uterine gestation.

Now, a few words with regard to the later months. As I have said, you have then to diagnose principally between extra- and intra-uterine gestation, not to make out whether pregnancy at all is present. You will remember that a case came up to the hospital for diagnosis last week. For five months the patient had suffered from amenorrhœa, although she had not been quite free from loss of blood for all that time. She had told the doctor who sent her up that she had menstruated once after the five months, that is to say, that she only had four months amenorrhœa. When we came to inquire into that carefully we found that she had her last regular menstruation five months age, but four months ago she had a very slight show of blood, which only lasted a short time. She has had one or two small losses since which, practically, do not amount to anything in the way of flooding. She had a tumour in the abdomen which occupied the right side, and had been fixed there for some weeks. She had constantly a great deal of pain on the right side. That was the whole history, and there was not any very strong evidence of extra-uterine gestation in it, though there was, undoubtedly, some suggestion. There was no doubt that there was a fœtus inside, for I felt it move when I examined her bi-manually. I felt a contraction in the tumour the first time I palpated her abdomen; but that was before I had questioned her, and I was rather expecting it to be a case of extra-uterine gestation, so I thought very likely I had been deceived.

I afterwards found out that the uterus undoubtedly did contract. There was then no question about the mass *not* being extra-uterine gestation, but being the gravid uterus.

With a little gentle pushing one could get the uterus, or this mass, we will call it for the present, right over to the opposite side of the pelvis. There was no difficulty in getting it into the middle line. That was absolutely against its

being a case of extra-uterine gestation, unless it happened that the fœtus had slipped after secondary rupture into the abdominal cavity and was free. Supposing the broad ligament with an intra-ligamentous pregnancy had constituted the mass, it would have been quite fixed.

She was five months pregnant, from her history, and if she had been five months pregnant with extra-uterine gestation it must have been broad ligament gestation, and there had been no secondary rupture. The fœtus was not moveable as a free body across the abdomen, but had to be moved inside the sac, in which contractions were felt. These two facts absolutely negatived extra-uterine gestation, so that there was no difficulty in saying definitely that she was not the subject of it; but that probably she had some perimetritis behind her uterus, and the growth of the uterus dragging on the adhesions caused pain; the adhesions also being the cause of the uterus remaining most to the right side. The case was, in fact, a further development of fig. 1.

With regard to treatment in any case of ectopic gestation, I do not think I need say much. As a matter of fact, it is, in principle, simplicity itself. Directly you make a diagnosis of extra-uterine gestation you must operate, whatever the condition of affairs is. I think it may be said that there is no exception to that rule. Formerly it was the custom to pass electric currents through the sac, to inject morphia into the sac and aspirate it, and do various other things; but now nobody thinks of doing that, even in the early months. In the later months it was supposed to be right to wait until the fœtus was dead, because there is always considerable difficulty with the placenta. In early tubal gestation it is easy enough to operate, as a rule, because you can remove the gravid tube entire. It is, however, more difficult when the ovum is more developed, because there is a great enlargement of vessels all the way round the placenta, and you have to cut through the placenta or detach it at the time you operate. This causes free hæmorrhage, which might be fatal in a short time if the separation of placenta is not very rapid, or if pressure cannot be at once applied. Even, in spite of this risk, it is a right thing to operate and remove the fœtus. Nowadays we are able to operate with far more certainty of being able to arrest hæmorrhage; or, if the placenta has to be left behind, of being able to keep the cavity aseptic.

CLINICAL NOTES ON CASES IN THE OUT-PATIENTS' WARD, LONDON HOSPITAL, DEC. 20, 1894.

BY

MR. JONATHAN HUTCHINSON, JUN.

A Case of Undescended Testicle on the right side, with a Hernia on the left, the Testicle below which is quite normal.

The emptiness of the right side of the scrotum is at once seen, and the testicle is felt with the epididymis just below the external ring. The testicle itself is not more than half an inch in its long axis; in other words it is extremely small, and may be described as undeveloped rather than as atrophied. The patient has never known it to come lower than it is now. He wore a truss from the age of 6 to 10, and an attempt might be made to ascribe the atrophy of the testicle partly to the pressure experienced during those four years from the truss. It is known, however, that for some obscure reason these retained testes do not develop properly: they are very rarely of normal size, and they are very liable to cease secreting when the man is between 20 and 30.

One of the most interesting features of the case is this: a man with a partially descended testicle has a tendency to hernia on the same side, and here for a certainty coming down over the testicle is an open processus vaginalis, reaching down as low or lower than the testicle, and hernia would be expected readily to form on that side. By a curious coincidence he has had no hernia on the right side, whereas he has a typical bubonocele on the left side, reaching down to the external ring. So great is the risk of hernia with these undescended testes, that it is advisable in some cases to excise the testicle in order to be able to sew up the canal. This case is exceptional, because the hernia has come down on the side on which the testicle has normally descended, and is of interest also from the extreme atrophy of the other testicle.

There is a specimen which I placed in the London Hospital museum, showing the testes undescended on both sides, lying just within the internal ring; on both sides the process of the peritoneum extends to the bottom of the scrotum. In one there was a hernia, in the other there was not. There may have been a hernia on both sides earlier, but at the time of death there was a large lump of omentum on one side only, reaching four inches below the testicle. In that case both testes were examined as to their secretion; spermatozoa were looked for and none were found. No great importance can be attached to this fact, since the subject had been dead for a week or two, so that spermatozoa might very easily be missed. Still, spermatozoa are very persistent, and may be recognized in stains on linen many days after they have been deposited there.

In the present instance is the man's risk of getting trouble from this non-developed and only partially descended testicle sufficient to justify operation? The only operation that would be justifiable would be the excision of the right testicle and closure of the process of the peritoneum.

The patient has never experienced any hernia on this side, and therefore it might be said that there is no need of operation. The testis is not in a prominent position, and he says he is not aware of ever having bruised it, in fact he has had no trouble from it. It is quite unlike a testicle in the perineum, which no doubt is always a nuisance to a man; for instance, he can hardly ride on horseback or on a bicycle without jamming the testicle between the saddle and the pubis.

This case is quite different; the man could ride a horse and also a bicycle without any trouble. A testicle just below the external ring does not appear to be any more subject to injury than if it hang down, and not so much. Supposing, however, he had gonorrhœa and gonorrhœal epididymitis, affecting the right testicle, there would be a chance of inflammation spreading to the peritoneal cavity. Some week or two previously a man with a retained testicle came to me with gonorrhœal epididymitis; fortunately for him it affected the testicle which was down and not the one up in the canal.

There is also the liability to grow sarcoma, for it is admitted that these retained testes are more prone to develop malignant growths, for some obscure reason, than normally descended ones; the risk is not very great, although it is a distinct point in favour of operating. There is no family history of tumours in this case, except that his mother's sister died from cancer.

There are three points in favour of operation: (1) the chance of his getting gonorrhœal epididymitis, affecting the retained organ, (2) the possible development of new growths, (3) the more important risk of hernia which, if it got strangulated,

might be very awkward to deal with. Against removal it is suggested that if the normal testicle came to grief, the retained one might, from pure benevolence, take to increasing in size and becoming useful. This is improbable; the retained testicle is atrophied and there is no likelihood of its ever growing larger. Such a testicle probably does not secrete spermatozoa and will never go on to development. Failure to come down is certainly not due to narrowness of the canal, because the testicle has got beyond that point. One operation that might be suggested is transplantation, pulling it down and fixing it to the bottom of the scrotum. The drawback is that when you get the testicle down and fixed to the bottom of the scrotum, the chief result is for the latter to be drawn up to near the ring. There is also the method of fixing a spring clip above to press the testis down after the operation. The patient wears the clip for three or four weeks with the view of squeezing the testicle down, but I do not think one can expect much from that.

A very ingenious suggestion of Mr. Keatley's is to make a raw surface on the thigh and to fix the testicle and scrotum to this, then after a year or so the adhesion is divided. This method is more likely to result in a successful issue than the use of the spring clip. It involves, however, a very great deal of trouble for only a very slight gain. I have done two or three of these operations of pulling down the testicle and fixing it in position. The chief gain has been the radical cure of the hernia where the latter has been present, and, in addition, the testicle has remained perhaps an inch lower down, but the ultimate result is not great. A horseshoe pad would be the proper sort of truss if any is to be worn, but the patient is too old to start that. I cannot imagine what sort of shield would really be worth his wearing, any stiff one would be very likely to favour a contusion, of which there is obviously no great risk. He has been very active and is going to sea again, but if ever there is any appearance of rupture on that side I should advise an operation. If not, I should let it remain, wearing a truss on the left side for the bubonocele.

A Case of Suture of the Ulnar Nerve after complete division about an inch above the Pisiform Bone.

This took place in December, 1893. I saw the patient first in March, 1894, and the nerve was sutured in April last, that is five months after the accident.

The case is of great interest from the very gradual way in which recovery has proceeded. Before the operation, there was complete anæsthesia of the front of half the ring finger and the little finger, but not of the back, because the division of the nerve was below the dorsal branch. Even in front the anæsthesia was not absolutely complete. There was great wasting of the abductor indicis, the first dorsal interosseous, the muscles of the little finger and the adductor pollicis. The two paralysed fingers were markedly colder than the others, it might be suggested that this was due to the division of the ulnar artery at the same time as the ulnar nerve, but, arguing from what one constantly sees in infantile paralysis, we may say that the coldness is probably due to the actual nerve division and the comparative disuse of the fingers. Then there was paralysis of the two inner lumbricales with the characteristic position of the fingers affected (most marked in the little finger), namely, slight hyper-extension of the metacarpo-phalangeal joints and flexion of the interphalangeal ones. He could not adduct or abduct the extended fingers in the slightest, owing to paralysis of the interossei.

When the ends of the nerve were exposed the upper end was found to be bulbous and the lower end somewhat tapering, with a gap of half-an-inch between the ends. It was necessary to cut off the rounded end of each so as to allow new nerve fibres to grow, leaving a gap of perhaps two-thirds of an inch. To overcome this, the wrist was flexed upon a splint and was kept thus for three weeks. The nerve ends were secured with catgut and fine silk, fearing that the catgut might soften too soon. He has now several very fine silk threads buried in the nerve. The same kind of silk is used in advancing the recti muscles of the eye, where the sutures are left buried under the conjunctiva, it never gives rise to trouble, and is easier to manipulate than catgut.

Recovery in this case was very slow, but is now almost complete. Sensation was almost completely restored at the end of a month, but the adductor and abductor movements did not return for about three months. The usual experience is that sensation is recovered before motion: it seems as though it were on a lower plane. The two inner fingers have straightened out and he can now abduct and adduct very fairly. Galvanism has

been steadily used. The muscles of the little finger are now well developed; they were completely wasted.

This case shows that one must not soon despair after the operation of nerve suture. After sewing the ends together, if you do not find improvement in two months, you think, perhaps, they have separated, and are, perhaps, disposed to attempt a further operation. No doubt, if the division has happened a good time previously, one must allow some months before the new nerve fibres grow down, and, therefore, must not be discouraged if recovery is not very rapid.

A most important point in such operations is to get primary union, for any marked suppuration is almost sure to spoil the result. Primary union must be attained, and, therefore, one cannot be too careful with antiseptics before and after the operation.

Extensive Alopecia Areata in Husband and Wife.

In this case (the male patient) the alopecia involves almost the whole scalp, and he is obliged to wear a wig. The wife is not reduced to the same painful necessity, but she has a patch of alopecia as big as the palm of the hand. In both husband and wife it has developed since last March. This points very strongly to alopecia being contagious. The man is 23 and the wife 22, and they have been married nearly four years. It is difficult or impossible to fit in such a case with any nerve influence; whereas it agrees very well with the idea of contagion, and one knows that every now and then a fungus is found actually in the form of very fine spore.

Another argument against the nervous origin is the frequent symmetry (rough, it is true) of alopecia areata. Most skin diseases due to nervous origin are non-symmetrical, whereas this is almost exactly symmetrical. Progress towards recovery in this case is slow, and always is so. It is only remarkable to find it advancing as much as it has done. Stimulating applications have been used, and the prognosis is not hopeless, there being good evidence that the hair bulbs are not destroyed, seeing that in many places fresh hairs have grown since he came under treatment. In the wife's case also the same result has followed.

It should be mentioned that in neither can any history of ringworm be obtained.

A Case of very extensive Goitre, symmetrical, and involving every part of the Gland; the two Lobes being involved equally with the Isthmus.

The neck measures $17\frac{1}{2}$ inches round at its greatest circumference. The isthmus of the gland can be felt reaching up, overlapping the thyroid cartilage, extending nearly down to the sternum, being quite two inches in depth. There appears to be no large cyst; the case is one of general enlargement, probably the result of a great number of small ones.

One point of interest is that the patient does not come from a goitrous district at all. He has lived for the last ten years at Forest Gate, whilst this has been forming, and there is no family history of goitre. It is a case quite unsuitable for excision, seeing that there is no part of the gland which is not involved. There is no urgent call for operation, the only inconvenience being that it gives rise to shortness of breath if he hurries at all, but there is no difficulty in swallowing. He is not at all anæmic, as many of the goitrous cases are, and his general health does not seem in any way affected.

A trial is being given to iodides of sodium and potassium—45 grains a day—not an excessive dose, and one which may be further increased before giving it up. The only operation to be suggested might be a division of the isthmus and a removal of part, with a view to prevent any pressure effects upon the trachea.

A Case of Tubercular Enlargement of the Left Knee-joint in a Syphilitic Patient.

Within the last sixteen months the man has had secondary syphilis, and has still throat ulceration from time to time, undoubtedly syphilitic. He has had great enlargement of the left knee-joint for four months, with all the characters of pulpy swelling. He was sent to the hospital with a diagnosis of syphilitic disease of the knee-joint. I feel quite certain that it is not so, but is ordinary tubercular disease of the knee-joint in a man who has chanced to have secondary syphilis at the same time. No doubt there is a close simulation occasionally of strumous disease by syphilitic arthritis, but it is always tertiary, and generally ten years or so after the primary disease. Cases are not found where, in the secondary stage within 18 months of infection, you get this condition, and

that is a very strong argument against the syphilitic theory in the present case.

The patient is a delicate subject, a man from appearance very likely to get tubercular disease, though he says has never heard of consumption in his family. Secondary disease of joints, and especially tertiary, yield pretty well to treatment, but this patient has had iodide pushed for many months without any advantage Secondary affections of joints are very temporary, disappearing, as rule, in a few weeks. The leg is, unfortunately, getting. more and more bent, but although the flexion has increased, the swelling has diminished, and is less angry looking than it was two months ago. The leg must be straightened. Gradual extension on an inclined plane will probably get it straight, but the question of excision or erosion of the joint must be entertained.

He has been sent to the seaside with the limb in a Thomas's splint, and, as already noted, a careful trial was given to iodide of potassium.

THERAPEUTICAL NOTES AND FORMULÆ.

Picrotoxin.—MM. Semmola and Gioffredi, of Naples, remarked upon the necessity, before administering an antisudorific, of thoroughly understanding the pathogeny of the secretory disturbance. If this be due to irritation of sudoriparous nerves, remedies such as agaricin, atropin, etc., should be given; but if it be due to central vasomotor paralysis, excitants, such as picrotoxin, should be administered. They reported the case of a woman, 43 years old, affected with hyperhidrosis consecutive to influenza, frequently occurring in combination with peripheral vasomotor disturbances, in the form of more or less extensive red patches. There was no functional or subjective alteration of the nervous system. Atropine, agaricine, camphor, camphoric acid, gallic acid, etc., were tried without success. Picrotoxin was then employed, and three days later the sweating began to diminish, and within eight days completely disappeared. The daily dose used was $\frac{1}{64}$ grain.

Laparotomy in perforation of gut in typhoid fever statistics up to date are approximately seventeen operations, with one recovery.

Treatment of Syphilis.—Prof. Lang employs the following formulæ:—

R Protoiodide of mercury ... gr.xxij
   Ext. opium   ...   ... gr.viiss
   Lanoline ...   . gr.xxij
   Sugar milk   ...   ... ʒi¼
Sufficient for 50 pills. Sig.: One to two per diem.

R Calomel ...   . gr.xxxvij
   Ext. opium   ...   ... gr.iv
   Lanoline ...   . gr.xxij
   Sugar milk   ...   ... gr.lv
Sufficient for 50 pills. Sig.: One to two a day.

R Corrosive sublimate   ... gr.iiiss
   Ext. opium   ...   ... gr.viiss
   Lanoline ...   . gr.xxij
   Sugar milk   ...   ... gr.lv
Sufficient for 50 pills. Sig.: One to two per diem.

If necessary, he also associates the use of arsenic or the iodide of potash with these formulæ; also in pill-form, according to the following formulæ:—

R Iodide potash   ...   ... ʒiiss
   Sugar milk   ...   ... ʒi¼
   Lanoline ...   . gr.xlv
Sufficient for 50 pills. Sig.: Three, ten, or fifteen a day.

R Arsenious acid ...   ... gr.viiss
   Sugar milk   ...   ... ʒi¾
   Lanoline ...   . gr.xlv
Sufficient for 100 pills.

The addition of lanoline, which melts at the temperature of the interior of the body, and the sugar of milk which easily dissolves in the gastro-intestinal juices, provide that no particles of these drugs pass through the digestive tract unaltered, so that one may be certain that the quantity of the drug ingested will be absorbed. If one will employ an injection, he warmly recommends the following:—

R Mercury.
   Lanoline, ana   ...   ... ʒj
   Olive oil ...   . ʒiss
Sig.: Inject five ccms. under the skin of the back every three to four days at the beginning, every five to eight days when the symptoms retrogress progressively, and every eight to fourteen days—not more than twice—when they have completely disappeared.

(*Gaz. Deg. Ospitali.*)

Treatment of Delirium Tremens.—In the *New York Medical Journal* Bellamy concludes an article as follows:—

1. Delirium was controlled with greater rapidity and safety by trional than by other hypnotics.

2. In the majority of cases a marked stimulant effect was observed, possibly on account of the methylic ethylic elements which enter into the composition of the drug.

3. On account of the low temperature noted in all cases, trional must possess antipyretic properties, thereby stimulating its allies of the phenol group.

4. It was always well borne by the stomach, and in one case was rapidly absorbed when administered per rectum.

5. No unpleasant after effects were observed, and in all cases recovery was speedy, with the exception of two.

Acute Sciatica.—Make the patient comfortable by relieving him of pain, giving, in mild cases, from 10 to 15 grains of phenacetin; when the pain is severe, inject morphine in sufficient quantity to thoroughly arrest it.

Convulsions—At the moment of the attack, place the patient in bed in a well-aired room and bathe the face with a wet cloth. Administer at once an injection of warm water containing a dessertspoonful of salt and two tablespoonfuls of table-oil. Give, every five minutes, a dessertspoonful of the following mixture:

R Strontium brom. ...	...	gr.xxxj
Musk	...	gr.iij
Syrup of chloral ...	...	f ʒv
Syrup of orange-flower water	...	f ʒj
Linden water ...	...	f ʒijss

If it cannot be given, administer the following sedative injection:—

R Musk	...	gr.iv
Chloral hydrate ...	...	gr.7¾
Camphor	...	gr.xvss
1 yolk of egg.		
Water	...	f ʒivss

Should the convulsions persist, give inhalations of ether or chloroform. Do not consider the attack has ended until the child has urinated. After the attack, keep the child in bed, imposing absolute quiet and darkness. Give only milk

as nourishment. Administer the following every hour, in dessertspoonful doses:—

R Syrup of codeine...	...	f ʒiiss
Syrup of hydrocyamus	...	f ʒv
Cherry-laurel water	...	f ʒiss¼
Linden water ...	...	f ʒiss¾

Seek carefully for the cause, and institute appropriate treatment.—(*Tribune Médicale.*)

REVIEWS.

Diseases of the Ear. By A. MARMADUKE SHEILD. (Cassell & Co.)

Published at 10s. 6d.

We have very recently reviewed Mark Hovell's "Treatise on Diseases of the Ear," and remarked then that its very completeness rendered it rather a book of reference for the library than a familiar companion of daily life; but the little book now before us supplies, we think, such a daily companion. It is very pleasantly written, and contains quite sufficient details of diagnosis and treatment to make it reliable in all the ordinary cases of ear trouble likely to be met with in general practice. Of the four coloured plates it contains, Nos. II. and III. are excellent illustrations of the external ear as seen with the aid of the otoscope. We confidently expect that a new edition will speedily be called for.

Urinary Surgery. By E. HURRY FENWICK. (Wright & Co., Bristol.)

Published at 4s.

This capital little book forms apparently one of a series of "Epitomes of Modern Surgical Progress." Although the surgery of the kidney occupies the greater part of the work, it might almost with equal propriety be termed an epitome of modern surgical progress in kidney troubles other than Bright's disease. It is accurate, and fully represents all shades of opinion, being, what its name implies, an epitome of the best recent renal work. It is cheap, and we can honestly recommend it not only to teachers but to all practitioners, and we congratulate both author and publishers on its appearance. A capital bibliography, containing no less than 303 references, concludes the book, and makes it really a valuable work of reference, although its pages number but 207.

On the DIFFERENTIAL DIAGNOSIS OF CEREBRAL TUMOURS,

WITH

SOME REMARKS ON TREATMENT.

A Paper read before the South-Western, West-Somerset, and Dorset Branches of the British Medical Association.

By VICTOR HORSLEY, F.R.S., F.R.C.S.,

Surgeon to University College Hospital,
and the National Hospital for the Paralysed and Epileptic.

In thanking the South-Western Branch of the British Medical Association for the honour they have done me in inviting me to deliver this address on the subject of cerebral tumour, I ought perhaps to explain that it seemed to me that the *technique* of operative treatment of the same was now so well understood as not to require much discussion, whereas the determination of the existence of a tumour of the cerebral hemispheres is always a matter involving much responsibility, and never wholly free from difficulties which, in some cases, appear to be insurmountable. Not only, therefore, is the differential diagnosis, from this point of view, of the first importance, but it becomes still more so when we consider that upon such a diagnosis hangs the probability of being able to relieve the patient by surgical measures.

Omitting anomalous cases, it is generally recognised that when certain classical symptoms are all present in the same subject, that the condition can hardly be other than that of a cerebral tumour.

These classical symptoms are optic neuritis, headache, and vomiting, epilepsy, stupor, and paralysis. Few could fail to recognise that tumour was present if witnessing this association of formidable symptoms. Nevertheless, there are cases in which meningitis, abscess, and even uræmic poisoning precisely imitate tumour in producing the same signs. I take these three chief causes of error, not because I have time to dwell upon the details of differentiation applying to each, but because they are the commonest among sources of confusion, and it is easy to point out one or two salient conditions upon which a safe

differential diagnosis can be constructed. In the first place, as regards meningitis, careful inquiry into the history and causation will, I think, unquestionably prevent a mistake being made, and I am not aware of any instance in which operation in a case of this nature has been undertaken wholly in error. In the second question, namely, that of abscess, there should be, of course, no confusion between an acute abscess and a tumor cerebri. The case, however, becomes different when the condition is chronic. A chronic abscess may, in all essentials, so closely resemble tumour as to make a final determination impossible until an exploratory operation is undertaken. This is not a matter of such moment as it might appear, because, in any case, the skull must be opened for the permanent relief of either condition. While speaking of abscess I wish, with your leave, to allude particularly to one symptom, viz.: the temperature. It seems to be almost universally accepted, in any case by way of belief, that an elevation of temperature is characteristic, and therefore to be expected, in sub-acute abscesses; whereas, as Dr. Wilks pointed out very many years ago, the exact contrary is the case, and that sub-normal temperature is the characteristic feature to be looked for. I dwell on this point because I have seen very troublesome mistakes arise by reason of its not being recognised as a fact.

Finally, uræmic poisoning, as an especial source of error in diagnosing cerebral tumour, has attracted some attention of late, particularly in France. Knowing the possibility of this condition occurring, and its precise mimicry of cerebral tumour, it is gratifying to feel certain, as we may do, that the microscopical and chemical tests of the urine in habitual use will reveal the presence of renal disease without any further trouble. In leaving now the question of the classical symptoms of cerebral tumour I cannot pass to the next part of my subject without adverting for a moment to the symptom of optic neuritis: firstly, from the point of view of the assistance in determination of localisation, and, secondly, because it may be present together with certain of the other symptoms mentioned in a degree that may often mislead. I will speak of it in the first place as a localising

symptom. Having been struck for some time with the apparent fact that the optic neuritis was more marked on the same side as the cerebral lesion, and being, at the same time, impressed with Dr. Jackson's teaching that, on the contrary, it is more usually exaggerated on the opposite side, I some years ago asked my friend and colleague, Mr. Marcus Gunn, to kindly measure for me the amount of swelling in different cases, and the result of these observations was to show beyond question that the view that the swelling was most marked on the side of the lesion was correct for the majority of cases, but that there were exceptional cases in which it was more marked upon the opposite side.

The second point as to its occurring as a source of confusion. A further condition under which this is possible, to which I now desire to draw your attention, is that of simple anæmia ; and a case which I have in mind which was treated at the Queen's Square Hospital certainly presented remarkable difficulties in the shape of diagnosis. The case was that of a young lady who suffered from headache of a very severe character, optic neuritis, sickness, and when she was first seen by Dr. Ferrier a certain degree of hemi-paresis was present. She was admitted into Queen's Square Hospital, and I was there asked to see her with a view to operation for abscess or tumour. When I saw her the hemi-paresis had, for the most part, cleared up, but there were other symptoms of a remarkable character showing themselves (twitchings of the trunk muscles, etc.) pointing to cerebellar lesion, and the most important of which, in connection with my present subject, was the existence of a very loud bruit localized strictly to a small area over the right cerebellar fossa, midway between the centre of the fossa and the lateral sinus. It appeared to me that the most reasonable diagnosis that could be formed was one of a highly vascular tumour situated in the region mentioned, and the anæmic condition of the patient was looked upon as being probably secondary rather than primary. In view of the tentative diagnosis made, I decided to ligature the chief arterial supply of the part before proceeding to attempt anything further, and I, therefore, tied the common carotid, and, at the same time, what I believed, at the time, to be the right vertebral artery. But the determination of this was rendered uncertain by what was taken, at the time of the operation, to be puncture of the pleura, e.g., the

characteristic noise, etc., was heard. However, the patient made an excellent recovery, the wound healing by immediate union and without the least constitutional disturbance. From the moment of ligature the bruit disappeared, as also the headache, optic neuritis, and the cerebellar symptoms, and the patient left the hospital without any further treatment than that which was adopted (after the above-mentioned improvement) to counteract the anæmia which was so prominent a feature in the case. She has remained well with the exception that some of the symptoms, e.g., the headache, slightly recurred, and disappeared again with treatment with arsenic. Although, of course, there might have been a vascular growth which was arrested by the ligature of the vessels, I am strongly inclined to think now that it may, after all, have only been a case of anæmia which thus constituted an error in this instance of differential diagnosis: and if this were so one would naturally regret (after the event) having ligatured the vessels, although the patient showed no inconvenience and only benefit resulted from this treatment.

So far I have dealt only with cases of fairly marked symptoms of disease. But what we seek to do is to be able to detect cerebral tumour when as yet it is in an elementary condition, for it is only by perfecting our knowledge and ability in this direction that we shall render it possible for surgery to more successfully intervene. The possibility of forming an early diagnosis depends on the character of the initial symptoms of a cerebral tumour. It remains now to examine what are the common initial symptoms. Briefly these are headache, epilepsy, and paralysis. I will now discuss them in some detail.

Headache. Headache in cerebral tumour must be referred under two divisions. Firstly, as to whether the headache is diffused; secondly, whether it is localized. Diffused headache, as a rule, is an earlier symptom in cerebral tumour than the localized form. In this connection it is worth delaying for a moment to discuss the subject of cranial tenderness. Cranial tenderness is frequently looked for in one of two ways, either by percussion of the head with a rubber hammer, or by pressing steadily on the head with the ball of the thumb over different parts of the skull *seriatim*. I think the percussion method ought to be given up. It is a painful procedure to many people even in health, and in cerebral tumour *a fortiori* more so, and the patient's answers are confused

by the unnecessary distress inflicted, whereas by the method of pressure exact localization can very often be confirmed. A good instance of this is shown in the photograph of a case in which marked local tenderness was present exactly over the tumour.

No complete reliance is ever to be placed on pain in the head alone as a determining symptom. Finally, it is worthy of note, too, that the headache in cerebral tumour is described by patients as being either constant or paroxysmal, the latter being the more common.

Epilepsy. Of all the initial symptoms of cerebral tumour, the epileptic convulsion is the most important, not only because it is a clear indication, but also because, for reasons to be stated directly, tumours causing the most characteristic forms of epilepsy are the more easily removed. In cerebral tumour the character of the convulsion may be various. Thus it may be general, and so simulating idiopathic epilepsy; secondly, it may be generalized, but preceded by a localized aura; thirdly, though generalized, it may also be commenced by localized muscular spasms; fourthly, it may be a typical Jacksonian fit, becoming in some cases more generalized, and in some followed by a certain degree of paralysis; and lastly, a cerebral tumour may evince itself by single spasms, not grouped as in a complete fit. To effect an analysis of the epileptic convulsion in cerebral tumour, it is necessary to have a clear conception of the topography of the different regions of the cortex of the brain, and their relations to different portions of skull. Therefore, no one should undertake operations of this kind without a very thorough study of this subject, a study which now, thanks to Professor Cunningham, of Trinity College, Dublin, is rendered so easy and exact by his very beautiful casts of dissections showing the brain *in situ*. Photographs of some of these I now show you to illustrate the striking difference there is between the new-born child and the adult in this particular. Having completed this portion of the subject of topography, the next question naturally is the position of the various foci or areas of representation, excitation of which produce what we call an epileptic fit. Probably you are familiar with the now classical diagrams of the position of these centres. However, I show you a photograph of the human brain taken from one of Professor Cunningham's models, on which I have marked the situation in the human subject of those

centres, the precise situation of which has been accurately determined in operations undertaken for the relief of focal epilepsy. You observe that they have the same relations to each other as in the orang. Further details I will allude to, as it may be necessary in my subsequent remarks.

I now wish to show you a series of cases to illustrate the next important point in connection with the topographical relations of the genesis of the epileptic convulsion, namely, an examination of the conditions under which we may hazard diagnosis of the seat of mischief from the observation of the character of the fit.

(*a*) *Frontal lobe.* Lesions of the frontal lobe appear to produce convulsions of the generalized type, and, above all, as Dr. Jackson has often pointed out, convulsions in which movements of a semi-purposive character are very prone to be exhibited. In a case I recently had under my care, in which, at the request of Dr. Beevor, I opened the skull for relief of pressure, and a case of which the further progress is now being watched by Dr. Shaw, of Maidstone, a further point of much importance in the epilepsy of tumours of the frontal lobe was very obvious. This is the fact, that in the fit the focus which commences the disturbance is that for the turning of the head and eyes to the opposite side. The consideration of the situation of the different foci of representation to which I have just drawn your attention explains the significance of this—namely, that as the disease spreads from before backwards, naturally the first focus to become involved is that for the movement in question.

(*b*) *Parietal lobe.* Turning to the parietal lobe, the first photograph exhibited is that of a fibroma of the cortex which I removed, the patient living nearly three years afterwards, there being reason to suppose from the fact of the patient dying from pachymeningitis that the lesion was a gumma. The diagnosis in this case was based on the fact that the patient had well-marked solitary spasms of the limbs on the right side, and I also mention the case to exemplify the desirability of treating gummata by operation as being incurable by drugs. But the commonest form of convulsion which is due to lesion of the parietal lobe is that which is justly known as Jacksonian epilepsy. It is hardly necessary for me to dwell upon the details of so well-known a condition, but having now had the opportunity of watching the first case of tumour that I operated upon eight years ago

from commencement to finish, the patient having recently died of tuberculosis, I wish to show you a series of photographs illustrating a typical case. In this case the patient, when admitted into Queen's Square Hospital, only exhibited Jacksonian epilepsy, and slight paresis of the special kind to be detailed presently. Having operated for the removal of tumour, I described the case and exhibited the patient at the Meeting of the Association at Brighton in 1887. The patient was relieved of his symptoms, although he had a few convulsions some months after he left the hospital, the causation of which was never made clear, and which never recurred. As just stated, he died a short time ago of general tuberculosis. You now see the tumour and the appearance of the brain as found at the post-mortem examination. The brain shows simply a small depressed scar strictly limited to the field of the operation, and, in accordance with the topography which we have already discussed, you will observe that the scar is situated exactly at the inferior genu of the fissure of Rolando.

(A second series of photographs were then shown of a case illustrating the recurrence, the successful removal of a large glio-sarcoma of the parietal lobe, and the subsequent recurrence of the same; and it was pointed out that, with the better knowledge of these conditions nowadays, considering the comatose condition of the patient at the time of the operation, that a curative operation would not be attempted in such conditions, but only a palliative one.)

(c) *Parieto-occipital region.* Two cases of lesion of this region having come under notice, and both presenting similar marked symptoms, it is probable that in all cases of tumour of this region the convulsions will be found in the main to be generalized, and accompanied by ocular deviation and visual auræ.

(d) *Occipital lobe.* Tumours of the occipital lobe most commonly present generalized convulsions, hemianopsia from destruction of the cuneal region, and are not infrequently accompanied by so-called hysterical manifestations. Hemianopsia, it is to be noted, is also a frequent, and, in fact, usual accompaniment of tumours of the parieto-occipital region, where the lesion burrows deeply, and so affects the optic radiations.

Finally, in the case of tumours exciting epilepsy from the occipital lobe, it is to be remembered that, owing to vertical pressure on the tentorium,

they may also give rise to symptoms resembling closely those of cerebellar growth, *e.g.*, nystagmus, tottering, etc.

(e) *Temporal lobe, outer surface.* The epileptic convulsions from lesion of the temporal lobe have been observed in cases of gross organic disease, published by Dr. Thomas Wilson and others, to be preceded by a sensory aura of an auditory type; also by the occurrence of amnesia, and further that where paresis follows that is apt to be of a graduated type from pressure on the pyramidal fibres and areas of "motor" representation in the cortex.

(f) *Temporal lobe, inner surface.* Those cases of tumour of the inner surface of the temporal lobe, which have been carefully observed, are extremely interesting, for they have shown, as described by Dr. Jackson, Dr. Beevor, and Dr. Anderson, that the epileptic convulsion is signalised by the occurrence of hallucinations of smell and taste, the special sense areas of representation of these functions having originally been demonstrated by Dr. Ferrier to be situated in this region.

I wish, after the foregoing brief summary, to next draw your attention to the characteristics of certain anomalous cases—that is, cases which are considered anomalous, because perhaps the notion that the character of the fit in a case of cerebral tumour must be the Jacksonian type has too easily gained general acceptance. In the first place it should be noted that whatever be the nature of the fits in the subsequent progress of the case, the initial attack is very often a generalized one. Not only is this the case, but also in a certain number of instances the attacks are sometimes localized and sometimes generalized in the same case. The specimen before you is the largest tumour I have ever removed, and which occurred in the case of a lady operated upon six months ago, she being still in good health, and recovering from the paresis to which I shall refer directly. The case was treated for more than nine years as one of idiopathic generalized epilepsy, and that even at a time when the growth was already penetrating the skull. A careful analysis and observation of the fits would have shown that many of them were characteristically unilateral. There were also other symptoms indicating cerebral tumour, which unfortunately escaped attention, but with these we are not now immediately concerned. In speaking of such mistakes, which may arise from the occurrence of generalized fits, there are two further

factors worthy of your notice, and which need only to be mentioned for you to recognise their importance. I refer to the position and size of the tumour respectively.

As regards position, there can, I think, be no doubt that the more deep-seated the growth the more generalized is the convulsion. And as regards its size, the larger the tumour the more generalized is its effect. Any such modification, therefore, of the character of the attacks will have special significance in considering the possibility of surgical treatment.

Finally, on this point, the clinical duration of the case may be longer than might be anticipated. There seems to be some reason for believing that cerebral neoplasms may grow steadily and very slowly for a time, and only later assume a more malignant character.

The next symptom of great importance is that of paralysis, and I will speak of loss of movement first. There is frequently present in cerebral tumour a general muscular weakness, which has frequently been confounded with simple neurasthenia, and sometimes so much so as to unfortunately lead to a mistaken diagnosis of hysteria, and not a few cases of this kind have from time to time been reported. Differentiation can only be accomplished by a very careful attention to the details of every case, and of course it goes almost without saying that where decision is very difficult the case ought to be provisionally regarded as one of organic disease.

Paralysis of movement when limited to one part or one side of the body is, of course, a symptom of great value, and it may arise, (a) by direct destruction or pressure upon the so-called motor or excitable frontal-parieto cortex, or (b) by pressure transmitted indirectly to the same part from a lesion situated at a distance, i.e., in some other lobe. Then, also, the loss of movement may be due to the fact of the tumour destroying the descending pyramidal fibres in either the corona radiata or the internal capsule. In the cases of such indirect interference the situation and degree of the paralysis is precisely dependent upon the situation and size of the growth, as is well shown in the photographs before you of circumscribed glioma of the right hemisphere in a case in which I refused to operate as being too far advanced.

As regards indirect pressure, i.e., from a lesion at a distance, I have long laid much stress on the necessity for looking out for the gradation of paralysis, inasmuch as this offers a clear guide to the situation of the lesion; e.g., in tumours of the temporal lobe, where the pressure is, of course, upwards, the paralysis is more marked in the face, less in the upper limb and more in the lower limb, these being the relative situations of the areas for each part. Similarly, in lesions of the parieto-occipital region the intensity of the paralysis is greatest according to whichever area of representation happens to be in the same horizontal level as the lesion.

Secondly, as regards paralysis of sensation. Here we enter upon very debatable ground; but I am satisfied that the view always held by Professor Hitzig, namely, that the so-called motor region is really sensori-motor is the correct one, and that we ought to adopt the earlier terminology of Dr. Bastian, and speak of the fronto-parietal region as kinæsthetic. In 1886 I thought I established the fact that in extremely limited lesions of definite spots in the so-called "motor" region in man there was present a certain degree of tactile anæsthesia, but chiefly, as Bastian and Hitzig had pointed out, that there was loss of muscular sense, and I showed reason for believing that the paralysis of sensation was proportional to destruction of the cortex, and had the same distribution in localization. I now show you two photographs of more recent cases of removal of cerebral tumour which I had last year at Queen's Square Hospital, and in one of which, thanks to the kindness of our president to-day and Dr. Gibson, I have the opportunity of showing you the patient this afternoon. These two cases, as you see from the photographs, control each other, inasmuch as, in the case before you, the lesion did not involve the face area as in the other instance, with the result that there was then none of the anæsthesia of the face present in the other case. I need not weary you with going over old ground, but only wish to demonstrate to you the fact that it is absolutely necessary, in looking for what may be called cortical anæsthesia, to employ the method I have always insisted upon, namely, the indication of the exact spot touched by the patient with his opposite hand, the stimulation being an extremely light one, and the patient's eyes, of course, screened. Accurate determination of the truth of representation of tactile sensation in the parietal cortex was recently made by Dr. Mott in

a valuable series of experiments in the monkey. This special combination of slight loss of tactile sense combined with loss of muscular sense, and which I hold to be characteristic of a parietal cortical lesion, is not to be confounded with the profound loss of tactile sense which occurs in lesions involving the posterior third of the internal capsule. Comparing the phenomena of paresis with those of epilepsy, we can, with perfect accuracy, make the general deduction that epilepsy is an early symptom, and paralysis a late indication of cerebral tumour.

Numerous other symptoms, of course, are well worthy of close analysis if time made it possible, the most prominent of which, of course, is the condition of the deep reflexes and the contrast between the well-known exaggeration of the reflexes on the opposite side of the body in cases of destructive cerebral lesion, and of a like phenomenon on the same side in cases of cerebellar mischief, as so recently established beyond doubt in the very complete researches of Dr. Risien Russell, and which have rendered this point as definite as any in the history of cerebral tumour. In speaking of cerebellar tumours even by way of parenthesis, I feel the paramount importance of reminding you that such lesions exert a distal effect on the cerebral hemispheres which may, unfortunately, lead to some grave error in diagnosis, and the coarse anatomy of which is well evidenced in this photograph, which shows how much tension and consequent dilatation is observed in the cerebral ventricles when cerebellar tumour interferes with the downward circulation of the cerebro-spinal fluid.

The error in diagnosis to which I have just referred is the possibility of being deceived as to the nature of the epilepsy in such attacks. Some time ago Dr. Buzzard asked me to operate in a case in which the symptoms pointed very strongly to a lesion of the left parietal lobe, a case in which, during my examination of the patient, the actual opportunity of observing a fit occurred. The patient was a small boy, who had all the symptoms of a parietal lobe cerebral tumour—namely, stupor, headache, vomiting, hemiplegia, intense optic neuritis, etc., and, apparently, Jacksonian epileptic fits becoming generalized. In addition, I found that he had nystagmus, and a history of reeling to one side. Under these circumstances I thought there might be two lesions, one sub-tentorial, but the other certainly in the parietal lobe. On operating for the latter, although the part was freely exposed and very thoroughly explored, no tumour was found, the bulging of the brain proving, however, that a deeply-seated tumour existed within the skull. However, I closed the wound, and, in accordance with what I published at the Congress at Berlin, and at the Meeting of the British Medical Association at Newcastle, release of the pressure caused arrest of the growth, and probably its complete atrophy, for all the symptoms described disappeared, and the boy is now apparently perfectly well, only a slight clumsiness in the hand being observable. It is clear that in a case of this kind one mistook the nature of the attack, which was probably a compound fit of cerebellar origin, but which by correlation of the cerebellum with the cerebral hemisphere obtained a misleading character.

In leaving this necessarily very imperfect summary of symptoms, there is one great principle which they all observe, and which serves to differentiate them from the great group of vascular lesions and their effect, namely, that during the weeks and months of the patient's illness the symptoms are, if not masked by treatment, steadily progressive, and observation of their steady extension should always be looked upon as the most pathognomonic sign possible.

Although this collection of rather scattered observations deals with the diagnosis of cerebral tumours, I must ask to be allowed to make a few observations on the generally received practice of treatment of the same at the present day. It is the custom to deal with cases presenting the symptoms I have described with drugs for many weeks, months, or even years, the patient steadily deteriorating the while. Six years ago I made a suggestion that all cases of Jacksonian epilepsy should be explored (provided, of course, the sources of error I have described this afternoon have been excluded), after six weeks' unsuccessful treatment with drugs; and by that I mean treatment which has failed to reduce these symptoms by half or at least two-thirds of their intensity. The only notice of this suggestion has been its adoption in principle by Dr. Allen Starr in his very valuable work on cerebral tumours, and in the recent very successful case of my colleague, Mr. Ballance, recently published by him, with Dr. Beevor. No doubt now, although a good many years after its proposal, this plan may receive

greater favour.   In addition, it seems to me that the present practice of waiting till optic neuritis has developed is unjustifiable.   We ought to anticipate the incidence of so grave a symptom.   But I would go further, and I would say that, to my mind, if we employed in ordinary practice the procedures which it is customary to use in the case of cerebral tumours, our practice would be considered contrary to medical ethics, because the treatment of such cases by drugs means the persistent application of useless remedies in a fatal disease to which the patient is obviously succumbing week by week.   To put it in another way, who would dream to attempt to justify himself if he treated a new growth, when it attacks a limb, with iodide of potassium and mercury instead of an early operation?   Each case, of course, must be treated on its own merits, and no one should advise operative treatment, in my opinion, in cases which present specially adverse circumstances ; e.g., unusual risk to life, risk of permanent damage to very important centres, such as the centre for speech, or cases of very rapidly growing malignant sarcomata or glioma, in which the tumour being diffuse there is no reasonable hope of successful result.   In hopeless cases I can only draw your attention to what I have published in regard to palliative operation, to insist further on the value of that procedure by stating that since my last statements on the point I have had two more cases in which arrest of the growth and disappearance of the symptoms has followed mere release of the pressure.

I have only one word to add as regards the *technique* of the operation, and it is that the experience of the last two years has proved to me that the method I proposed of doing the operation in two stages, in order to avoid the shock which used to be such a source of danger, is adequate for the purpose for which it is desired, and I do not think any case now should terminate fatally from this cause.

In conclusion, I show the photographs of a case of a patient (Mr. G.), in which this mode of death happened, and who had been treated with drugs for many years.   The growth was one springing from the dura mater, and if recourse to surgical treatment had been made earlier, no operation would have been more easy or successful—a fact which has been evidenced by this result being obtained by me in a precisely similar case referred to me at Queen's Square Hospital by Dr. Bastian,

A POST-GRADUATE LECTURE
ON
CONDITIONS WHICH MAY BE MISTAKEN FOR IDIOPATHIC ANÆMIA.

Delivered at Paddington Infirmary,

By THOMAS D. SAVILL, M.D. Lond.,

Formerly Medical Superintendent of the Infirmary.

GENTLEMEN,—By the chances of clinical work, we have recently had in the wards of the Infirmary several examples of morbid conditions which are sometimes mistaken for idiopathic anæmia ; * and as I am able to show you to-day post-mortem specimens of some of them, side by side with their clinical histories, it will be interesting for us to study them together.   I have frequently, as you will see directly, fallen into such an error myself, and I have known others do the same.   The difficulties of distinction are greater than they would seem at first sight ; but the differentiation is of extreme importance, not only from the point of view of prognosis, but still more so of treatment.   It not infrequently happens that the first thing which excites the physician's suspicion is, that the case *does not yield to treatment by iron* after a fair and proper trial of its different forms.   At any rate, the anæmic condition in the diseases about to be mentioned does resist treatment by iron, and in some of the cases it was tried without avail.   There are, as of course you are well aware, some cases of true idiopathic anæmia which do not benefit by the administration of that drug, either on account of the patient's unhygienic mode of life, or for some other reason.   But they are not a large number, and it is not my purpose now to enter on that question.

I. *Latent Cancer, and especially Cancer of the Stomach.*   The stomach now shown to you is that of a woman, Charlotte Nash, aged 30, who was admitted on July 23rd for intense anæmia.   She suffered for twelve months from progressive weakness and pallor, and from amenorrhœa for six months.   On admission, she did not present the sallowness and cachexia of cancer, and none of
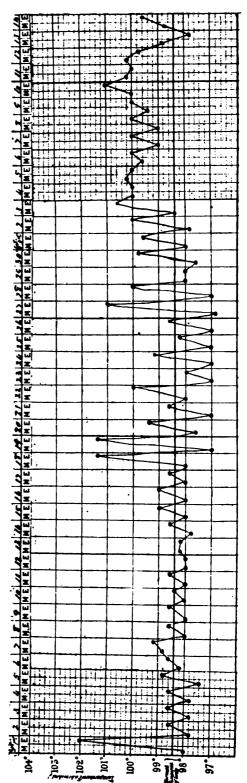
* That is, primary or idiopathic chlorosis, as distinguished from chlorosis or anæmia, secondary to hæmorrhage, or to other diseases in which it usually constitutes only one of the symptoms.

the physical signs; but the pallor and bloodlessness of her skin were extreme. There was no history of hæmorrhages and none of vomiting. A thorough examination of every organ failed to detect any physical signs. In spite of iron administered in many different forms she became weaker and still paler; and after September 2nd she had occasional vomiting of food for a few days, but never any blood or coffee-coloured vomit. The sickness quite ceased on September 12th, but she got greatly weaker, and died on October 23rd, without any physical signs having manifested themselves at our repeated examinations.

You will see that her stomach is the seat of a large mass of encephaloid carcinoma, towards the pyloric end, which, having been drawn up by adhesion to the under surface of the liver, had escaped examination during life. There were commencing secondary growths in the lungs, but the other organs were normal. Subsequently we discovered from her relatives that her father, mother, and one sister had died of " cancer."

Curiously enough about the same time a woman named Jessie Hopkins, aged 47, was admitted for what was thought at first to be idiopathic anæmia. In her case cancer of the stomach was suspected shortly before death, but for a long time it escaped detection, and even then the diagnosis was conjectural, owing to the absence of characteristic pain and vomiting. She died on October 14th, and a deposit of scirrhus was found in the pylorus.

Several other similar cases could be mentioned, showing that the means of readily identifying this condition are not invariably present. When the patient is advanced in years, has a typical cachexia, a sallow colour of skin, sunken eyes, and coffee-ground vomiting; or when there is a tumour in the region of the stomach, with pain, hæmorrhage, and fœtid discharge from the vagina or elsewhere (the four classical signs of malignant disease), the diagnosis is simple enough. But our first patient was only 30, and I have met with cancer of the stomach in a young woman only 28 years old. In such cases it is only by careful and repeated examinations of the stomach and uterus, by exhaustive inquiry into the family and previous history, and the failure of iron to relieve the condition, that a clue can be obtained. Nevertheless, it is extremely important, not only for purposes of prognosis, but because by the resources of modern surgery one might sometimes be enabled to prolong life.

CHARLOTTE NASH.

It is worthy of note, in passing, that the first of these two cases of cancer of the stomach had an intermitting pyrexia almost throughout. This is, I believe, a very unusual symptom in the disease; and the presence of a loud cardiac murmur (of hæmic origin, as we were able to confirm at the post-mortem examination) might have induced us to class the case as one of "pernicious anæmia," had not an autopsy been made. Her morning temperature throughout was normal or subnormal. During July and August her evening temperature sometimes rose to 99.5°, though ordinarily it was normal at night and subnormal (97°) in the morning. In the latter part of September and in October the evening temperature often rose to 100° or 101°.

II. *Latent tuberculosis.* That great clinical observer, Trousseau, used to say, "if you have a case of anæmia which resists treatment by iron, always suspect tubercle or syphilis." And there is no doubt that tubercle may be deposited in the lungs and other parts of the body, and be attended by considerable anæmia, yet it may remain for a long time without any physical signs, or even without any very grave disturbance of general health. You have seen a girl now in the wards, Louisa B., 22 years of age, who came in last May for a ruptured perineum. She also had intermitting pyrexia, which was at first attributed to this cause. The very anæmic condition of this girl for a long time puzzled us, and resisted treatment by iron administered in different forms. Her chest has been carefully examined many times, but it is only quite recently, after a preliminary period of seven months, that she has developed very slight signs in the lungs, which we now know to be due to the presence of tubercle, by reason of the bacilli which Mr. Wainwright has identified in the sputum.

Other cases could be mentioned, but instances of latent tubercle, giving rise to anæmia, are, no doubt, quite familiar to you. The anæmia of latent tubercle is indistinguishable in appearance from idiopathic chlorosis unless the hectic flush of the former be present on the cheeks. Both diseases are common, both arise most frequently at the same period of life, and both are apt to occur in consumptive families. The physical signs of tubercle may be absent, especially if only the deeper parts of the lungs be attacked; and it is only by the detection of bacilli in the sputum, or

the diligent use of the thermometer, that the two may be distinguished. In idiopathic anæmia the temperature is generally normal or sub-normal, but it may be taken almost as an axiom in medicine that an active tuberculosis never exists in any part of the body, even in the absence of physical signs, without causing an elevation of temperature. The pyrexia, moreover, is very characteristic. It is of a daily intermitting type, ranging generally from normal in the morning to 100° or 103° in the evening. I have recently seen, in consultation, a young man whose only sign or symptom for ten weeks was such a temperature, but at the end of that time he showed symptoms of tubercular meningitis, of which he finally died.

III. *Syphilis, either in the secondary or tertiary stage.* You have seen in the wards a girl, named Annie K., aged 20, who came in with anæmia. In her the ulcers on the legs are sufficiently characteristic, and she admits having contracted primary syphilis two or three years ago. You will have observed how extremely anæmic she is, and the cause is sufficiently obvious. The pallor is rapidly disappearing under the administration of mercury.

But the evidences of syphilis are not always so easily obtainable. On the table are microscopical specimens of a syphilitic gumma. It is a beautiful specimen of a growth of this kind (granuloma). This gumma was taken from the meninges of a lad, named Arthur L., whom I frequently saw in the Croydon Hospital many years ago by the courtesy of the late Dr. Lanchester, who gave me permission to publish the particulars of his case. He was 15 years of age, a grocer's assistant, of a most respectable family. His father was strong and healthy, his mother had died of phthisis, but he had five older brothers and sisters quite healthy. He himself had been quite well until ten months before, when he had begun to get pale and to lose strength—symptoms which had increased very much of late; he had also become a little deaf with one ear. He was admitted to hospital on August 5th, 1882, for very intense chlorosis, excessive weakness, and palpitation of the heart. He also had a cardiac murmur, having the characters which hæmic murmurs have. The lungs, abdominal organs, and urine were normal. He got gradually weaker and weaker in spite of the different iron preparations which were administered. The temperature went up a little every evening to 100° or 101°, and down again to normal in the morning,

all the time he was in hospital.* It was thought he might be suffering from "pernicious anæmia," or, perhaps, from latent tubercle. Once he had transient convergent strabismus, but otherwise no fresh symptoms presented themselves, and he died of weakness on September 7th, the mind remaining clear till the end.

At the autopsy the thoracic and abdominal viscera were examined without any result whatever ; but when the head was opened we found a yellowish gumma, measuring about ⅜ of an inch in thickness, and 2 inches long, by 1½ inches broad, stretching from before backwards, in the frontal region, on both sides of falx major, attached to the inner surface of the dura mater. Another smaller gumma was found in the occipital region, and another involving the petrous bone. All three were close to and narrowing some of the principal venous sinuses of the brain. After discovering these, we did what it must be confessed we ought to have more carefully done during life—examined the eyes, and found striæ in the corneæ ; and, carefully feeling the bones, we discovered gummatous nodules on the ends of some of the ribs and on the left clavicle. The inner surface of the principal arteries and the endocardium were of a deep crimson colour, the endothelium appeared to be thickened and covered by a thin layer of translucent material like red currant jelly.

The first of these cases was late secondary, or perhaps tertiary syphilis ; but the second case seemed to be in all probability one of hereditary syphilis, though no history could be made out. The anæmia due to syphilis can only be recognised by a history of the primary disease, or by any concurrent evidences of the malady which may be present, such as opacities in the cornea, or striæ in the lens, and an examination of the bones for nodes ; and finally by the circumstance that whereas iron altogether fails to relieve the pallor of syphilis, mercury will generally bring back the colour to the skin in course of time.

IV. *Lardaceous disease of the viscera* is another condition which might be mistaken temporarily for idiopathic anæmia. You have had several opportunities of seeing cases where the kidney was the main organ affected with this change.

But the translucent appearance of the skin, and a history of one of the two causes of waxy degeneration of the organs, namely, prolonged suppuration or syphilis, generally suggest the real condition with which we have to do. And the occurrence of a large quantity of albumen in the urine, or profuse diarrhœa, settles the question without any difficulty in well-marked cases.

V. *The pallor of Bright's Disease* may occasionally be mistaken temporarily for idiopathic anæmia. If there is any disease for which an infirmary offers a field for study, it certainly is the various forms of renal disease. You have, in my former lectures, seen many examples of these, and as the time is now drawing to a close I will only ask you to observe the characteristics of renal pallor in the numerous cases still in the wards ; and to remember that an examination of the urine affords us a ready means of diagnosing it from simple anæmia ; and also the age at which it generally occurs, in the case of the granular or so-called gouty kidney.

VI. *The anæmia of malarial affections* we not infrequently see in these wards, but such cases are always attended by a more or less general deposit of pigment in the skin, and especially around the eyes.

These are the chief conditions one has to differentiate from the primary chlorosis so frequent in girls. But it is to the first three conditions which I desire to direct your attention as liable to be confused in practice with idiopathic anæmia. In my experience in the wards, and *in the post-mortem room*, I have been impressed with the great difficulty of detecting carcinoma of the stomach during life, and especially when a slowly growing scirrhus attacks the pyloric end, and when, maybe, this organ becomes adherent to the under surface of the liver, and also in other instances where the classical signs are absent at any rate for some time.

I am further inclined to believe that a certain number of cases which are pronounced to be "pernicious anæmia" during life, and which are not examined after death, would have been found to be cases either of tertiary syphilis or *cancer of the stomach*. In each of these diseases there may be, as you have seen, a cardiac murmur and a pyrexia of a more or less intermittent type, just

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* Dr. Bristowe has published a case of gumma of the liver, attended by intermitting pyrexia, in the Clin. Soc. Trans.

as occur in "pernicious anæmia;" and that there may be an absence of the signs and symptoms which usually characterise the three conditions named; at any rate they may be so slight as to be easily overlooked. It would be well, therefore, to bear these facts in mind when in presence of a case supposed to be pernicious anæmia.

Gentlemen, we sometimes learn more by our own and other people's mistakes than we do by our successes. This is one of the reasons why I have ventured to bring these cases before you to-day. You will have seen how difficult it is sometimes to detect the conditions referred to, but being on the look-out for them, one is less likely to overlook them; and I believe you will often find that such cases are, or have been, perhaps during a period of many months, regarded by their physicians as cases of primary or idiopathic anæmia, and have been treated by iron on that supposition, but without success.

A CLINICAL LECTURE

ON

SEROUS PERIMETRITIS.

Delivered at the London Hospital, Jan. 14, 1895, by

G. ERNEST HERMAN, M.B. Lond., F.R.O.P.,

Senior Obstetric Physician to the Hospital, &c., &c.

THE author first related some cases in which serous perimetritis had been taken for an ovarian cyst. In one case he had been called to a medical man's wife, who was suffering from general peritonitis. As the symptoms of this subsided a large fluctuating swelling became palpable on one side of the abdomen. He took it for an ovarian tumour. The error was the more easily fallen into because her husband had thought he felt a swelling there before her illness. One of the most renowned ovariotomists in London saw the case in consultation with Dr. Herman, agreed in the diagnosis, and recommended operation. The operation was postponed, for reasons unconnected with the diagnosis, and in the interval the belly got smaller and softer, and the swelling finally disappeared. In another case he had been asked by a medical man to see and take into hospital for operation a case of ovarian tumour. Dr. Herman

saw the case, concurred in the diagnosis, admitted the patient into hospital, and fixed a day for operation. The patient was put on the table and anæsthetized. But he then found the tumour so much softer and less defined that he decided not to operate, and sent the patient back to bed. The swelling of the belly went quite away. He next referred to cases published by others in books and periodicals, in which the same mistake had been made, and even the abdomen had been opened, and serous perimetritis found instead of the ovarian tumour which had been supposed to be present. The lecturer then said:—

It is common in pelvic peritonitis for little spaces between adhesions to contain serous fluid. If we were obliged to use terms literally, we might call this "serous perimetritis." But in such cases the adhesions are the more important effect of inflammation: a few teaspoonful of serous fluid are of no consequence. Such cases we call *adhesive perimetritis*. We mean by "*serous perimetritis*" cases in which the serous fluid effused is important by reason of its bulk.

How serous perimetritis is produced. We know nothing of the special causes which produce, in a few cases, large exudation of serum, any more than we know why one pleurisy leads to adhesions and another to great pleuritic effusion. Perimetritis from any cause may be serous. Abscess of the ovary has been observed to cause it. The lymph that is first poured out becomes organized into fibrous adhesions, and then serous fluid is poured out under pressure, filling the space bounded by adhesions, and stretching tensely the parts which bound it. Why is so much serous fluid poured out? This pathological question is interesting, but at present unanswerable. The serous effusion is not a mechanical transudation, like the dropsy of heart disease, for the collection of fluid is so tense, and so displaces parts, that in the later stages of the disease it must be exuded against pressure. The transudation of much serum probably aids, in some way, the struggle of the phagocytes against the microbes, but we do not know how.
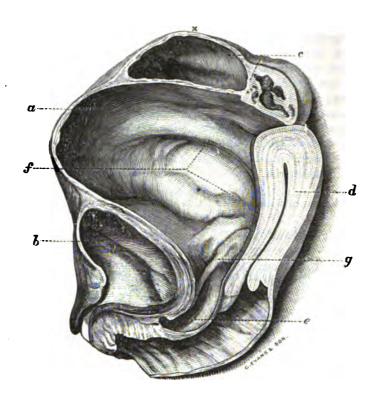
Special features of serous perimetritis. Serous perimetritis is important for two reasons: (1) Because it causes difficulty in diagnosis, and (2) because it leads to pressure symptoms. The collection of serum may be either (a) above the pelvis or (b) in the pelvic cavity. The accumulation of serum (a) above the pelvis more often

leads to diagnostic difficulties; that (*b*) in the pelvic cavity—*i.e.* in Douglas's pouch—more often causes pressure symptoms.

1. *Diagnosis between serous perimetritis and ovarian cyst.* When fluid is under tension, its reacting pressure tends to make the cavity which contains it round; for the sphere is the shape which contains the largest contents in the smallest periphery. Hence serous perimetritis forms a

often been advised for tumours which proved to be serous perimetritis.

The points in diagnosis are these: (1) In serous perimetritis the formation of the tumour is preceded by symptoms of inflammation—pelvic pain and fever. Small ovarian tumours cause neither pain or fever. (2) The tumour formed by serous perimetritis is fixed; a small ovarian tumour is moveable. (3) The tumour of serous perimetritis

DRAWING FROM NATURE OF A PERIMETRIC EFFUSION IN FRONT OF AND ABOVE UTERUS.
(*By permission of* Dr. W. S. A. GRIFFITH.)

a Cavity.	*e* Fluid pressing down between uterus and bladder.
b Bladder.	*f* Broad ligament.
c Cystic ovary.	*g* Ureter.
d Uterus.	*x* Point where Fallopian tube was adherent.

round fluctuating swelling, which may be either above or behind the uterus, but is always close to it. Now, the most common round fluctuating tumour near the uterus is an ovarian cyst. Hence serous perimetritis has often been taken for an ovarian cyst. It is especially likely to be taken for an ovarian cyst if it is above the uterus, and not in the pelvic cavity; but it may, when in the pelvis, be mistaken for a cyst. Operation has

is usually bounded in front by coils of bowel matted by adhesions; therefore it is generally resonant on percussion, while an ovarian tumour is dull. Attention to these points will, in most cases, prevent serous perimetritis being mistaken for an ovarian tumour.

Complex cases. But cases occur in which it is impossible to immediately say whether a pelvic tumour is ovarian or due to serous perimetritis.

Perimetritis may occur in a patient who has an ovarian tumour. Then the tumour will be fixed; there will be a history of pain and fever; bowel may become adherent in front of the tumour, or the contents of the tumour may decompose and liberate pus, and the tumour will then become resonant in front. In such a complex case the only criterion is the effect of treatment. Keep the patient in bed for a fortnight, and let the abdomen be painted daily with tr. iodi. If the tumour is ovarian, no difference will be produced; if it be serous perimetritis, at the end of a fortnight of this treatment it will be softer and smaller.

This diagnostic difficulty is of some historical importance, for probably some, at least, of the cases recorded by old writers in which ovarian tumours were thought to have been cured without operation, were cases of serous perimetritis.

Diagnosis of serous perimetritis from hæmatocele and from abscess. A serous effusion, an effusion of blood, and a collection of pus in Douglas's pouch, present the same physical signs. An effusion of blood into the peritoneum very seldom forms a tumour so large and tense as that sometimes seen in serous perimetritis; for in great intra-peritoneal bleeding the blood lies free among the bowels, and does not form a circumscribed tumour; and intra-peritoneal hæmorrhage small enough to be limited to the pelvis does not greatly displace the uterus or form a tense tumour. Hæmorrhage into the cellular tissue underneath the peritoneum forms a tumour, having the character of a parametric swelling, not of an intra-peritoneal effusion.

The diagnosis between a retro-uterine intra-peritoneal swelling containing *blood*, and one containing *serum*, is made by the history. Serous perimetritis is preceded by fever and pain; internal hæmorrhage comes on suddenly with faintness and pallor. An *abscess* behind the uterus, whether in the peritoneal cavity, the tube, or the ovary, if large enough to form a tumour like that of serous perimetritis, can only be distinguished from it by (*a*) its clinical course, which is that it shows no tendency to absorption; and (*b*) by puncture. Abscess behind the uterus is commoner than serous effusion.

Coagulum in serous perimetritis. Very rarely the serous effusion contains a fibrinous coagulum. Then the tumour will not distinctly fluctuate, but will feel solid or nodular. The possibility of some tumour other than an ovarian cyst will suggest

itself. The diagnosis of this rare form of serous perimetritis cannot be made until the cavity containing the coagulum has been opened.

Pressure symptoms. The tumour of serous perimetritis is *fixed* in the pelvis, and the fluid is under tension. It may cause (*a*) protrusion of the posterior vaginal wall at the vulva, by pressing Douglas's pouch down; (*b*) retention of urine, by driving the uterus so forward that it compresses the urethra; (*c*) painful and difficult defæcation, and even obstruction of the bowels, by its pressure on the rectum; (*d*) elongation of the cervix by stretching over the front of the tumour, and (*e*) sloughing of parts of the cervix, vagina or rectum.*

The course of serous perimetritis. Inflammation which leads to large and serous effusion is of a severer kind than that which ends in adhesions—that is, there is greater fever, greater prostration, and the disease lasts longer. Serous perimetritis is sometimes fatal. Its termination greatly depends on treatment. If pressure symptoms are relieved, and, if in relieving them, antiseptic care is taken, dangerous effects will be averted. Remember that the serous effusion is an *effect* of the inflammation, and indicates that the stress of the disease is past and gone, the invading microbes have been defeated, and the work of the organism is to repair the damage they have done.

Treatment of serous perimetritis. So long as the tumour is not large enough to cause pressure symptoms, no treatment other than medical treatment is required. Under such treatment (the chief thing in which is rest in bed) the serum will be absorbed and the tumour disappear.

If pressure symptoms are urgent, the size of the tumour must be lessened by removing the fluid. Even if you are in doubt as to the nature of the tumour, as it is fixed in Douglas's pouch (and serous perimetritis does not cause pressure symptoms unless it is within the pelvis), no greater harm can be done by puncturing it here than by opening the abdomen. You may tap it with a fine trocar, or aspirate it. I think the former better, because the strong suction of the aspirator more quickly withdraws fluid, and causes greater disturbance of the parts. The advantage of using a fine needle is that there is less danger of wounding

* For evidence of these and former statements, see Williams, "Obst. Trans.," vol. xxvii.; Routh, "Obst. Trans.," vol. xxviii.; Griffith, "St. Bart. Hosp. Reports," vols. xvi. and xviii.; Duncan, "Obst. Journal," 1878, and *On Perimetritis and Parametritis*: Doran, "Obst. Trans," vols. xxxi. and xxxiii.

a vessel in inserting it. The disadvantages are (1) that the only information which either trocar or aspirator gives is the nature of the fluid inside the tumour; and (2) that if the tumour contains fibrinous coagula, they will not be got rid of. I think it is better to cut through the posterior vaginal fornix with scissors, to an extent enough to admit two fingers, then to cautiously make a small hole in the wall of the tumour, and enlarge this by tearing first with one finger, then with two. Thus all fluid and clot can be withdrawn. Then examine the interior of the cavity, empty it, and either put in a drainage tube or pack the cavity loosely with iodoform gauze, letting the end of the gauze hang down in the vagina. Daily wash out the cavity with a non-poisonous antiseptic. It will gradually close.

When serous perimetritic exudation is situated above the pelvis it does not cause pressure symptoms, and medical treatment is all that is required; rest in bed with laxatives and counter-irritation if there be pain.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

January 22nd, 1895 :—

Simulation of Osteitis Deformans in Congenital Syphilis.

The subject of this case was a boy, aged 10, who had been sent up from the London Hospital by Mr. Waren Tay. There were no facts in the family history in any way corroborating the suspicion of syphilis. The father denied having ever had the disease, and there were three younger children who were healthy. It was stated that the patient had displayed no infantile symptoms, nor did his physiognomy or teeth suggest anything. However, notwithstanding this remarkable absence of corroborative facts, it seemed impossible to doubt a syphilitic taint. Both the boy's tibiæ were very much enlarged in all dimensions, and through almost their entire length, giving to the legs the appearance of being bowed forwards. The ulna in both arms was enlarged and thickened in a similar manner, and, as the radius had entirely

escaped, the forearm presented a peculiar, bent contour. There was also great thickening of the right humerus, and to a less extent of the left. The left femur a little above the knee-joint could also be felt to be thickened. The deformities produced were very remarkable, and exactly resembled those occurring in osteitis deformans in the adult. The thickening of bones had been first noticed about two years ago, and were stated to have begun in the right leg. There was little or no tenderness about the bones, but there had been much aching. In the legs the fibulæ, and in the forearms the radii, had, so far as could be ascertained, entirely escaped. (For comparison, a portrait, from which the first plate in the Illustrations of Clinical Surgery was copied, was produced. It was seen to represent, so far as the right humerus was concerned, almost exactly the same kind and extent of thickening as that present in the patient.)

Comments. This is a most remarkable case. I do not know that I ever saw more extensive bone disease in a child, and I never saw such an exact simulation of osteitis deformans. One peculiar feature of the case is the symmetry of the changes. When, many years ago, I drew attention to the frequent occurrence of enlargement of the tibia in connection with inherited syphilis, and to the fact that whilst it increased in size it also grew in length, I was able, in a number of cases, to prove the latter fact by comparative measurements of the two limbs. In this case, however, the two are exactly alike, and we can only infer that the affected bones are overgrown by comparing the leg with the thigh, and the whole lower extremity with the trunk. You will ask why, in the absence of any corroborative facts, I still insist on the diagnosis of inherited syphilis. My answer is that I have never seen bone disease of this character, in a child, excepting in connection with that cause. I have seen many in which the history corroborated the diagnosis; indeed, this is almost the first in which it was wanting; nor must we allow the absence of history to go for much. Many children are, probably, really the subjects of inherited syphilis, who do not show any signs of it either in infancy or in after-life. You will note that the child is the first-born of the family. It is very common for the eldest child to suffer and for the rest of the family to be healthy. Lastly, we may note that this is one of the cases in which a conclusive diagnosis may yet be given by future

events. The boy may have an attack of interstitial keratitis, or of deafness, which may set the question completely at rest. In the meantime we may observe that the osseous system is very extensively and severely affected, whilst no other structure either shows, or has shown, any evidence of taint.

Permanent Paralysis of Occipito-frontalis, with tendency to shed tears when eating.

A man, aged about 27, attended in order to demonstrate the fact that his left occipito-frontalis was entirely paralyzed. He could corrugate his eyebrow, and could use all the muscles of his face, but he could not produce the slightest transverse wrinkling of that side of his forehead. When at rest no peculiarity was observed in his face, but when animated or laughing it was seen that the left side of his face, as a whole, was a little wanting in expression. He could not close the left eyelids without screwing up the whole face. The history was that he had had an attack of very complete facial paralysis five years ago, from which, as he considered, he had wholly recovered, with the exception that it had left him deaf, to a considerable extent, in the left ear. He could just manage to hear a watch at a distance of about half an inch. A novel and very remarkable point in the case was that ever since this attack he had been unable to masticate food without shedding tears from the left eye. When his salivary glands acted, his lachrymal gland acted with them. This had been a great inconvenience to him.

Comments. I do not think that this case can be classed as one of the ordinary type of Bell's paralysis. In the first place, deafness attended it, which is not usual, and, in the next, the patient tells us that he had a very severe pain in the top of his head during the week that the paralysis first occurred. The patient is a little uncertain in his dates, but he admits having had syphilis about the time of the attack of facial paralysis, and I am much inclined to suspect that both it and the deafness were in connection with that cause. Attacks of syphilitic deafness, depending probably on inflammation of the fibrous structures in the internal and middle ear, are not very uncommon, and a very severe pain in the head is a frequent concomitant. Facial paralysis is rare in such cases, but a few examples of it are on record. The

affection is probably a form of inflammation of the structures composing the sense-capsule, which has its analogue in cases of syphilitic iritis. In some of the latter the inflammation is severe, involves many structures, and may destroy the eye, whilst in others it is slight and transitory. It is just so in syphilitic deafness. The permanent paralysis of the occipito-frontalis, and the defective power of inhibition of the lachrymal gland, are features in the case which should claim our thoughtful investigation. Nothing in my range of knowledge of the anatomy and physiology of the structures involved permits me to offer any plausible explanation.

If any of those present have under their observation cases in which complete recovery is supposed to have taken place after Bell's paralysis, it would be well worth while to examine them carefully, in order to ascertain whether any trace of conditions be present similar to those in our patient.

Psoriasis in a Child and Eczema in the Mother—Transmutation in Hereditary Transmission.

A girl, aged 12, brought by Dr. Conner, offered an example of psoriasis in an early stage. The patches were as yet but few in number, but were in their characteristic positions, and about the diagnosis there could be no doubt. They had been present about six months. The interesting point was that the mother, who came with her, was also the subject of skin disease. In her, however, it took the form of a chronic eczema of the fronts of the legs, with eczematous irritation at the roots of the nails.

Comments. I am interested in showing you these patients, although they are examples of common diseases, because they illustrate the law of transmutation in hereditary transmission. You will notice that the mother's patches of dry eczema are in almost exactly the same positions as the patches of psoriasis in her daughter. In both the disease is a chronic one, but no one would dream of giving to the mother's eruption any other name than that of eczema, or to the daughter's any other than that of psoriasis. The mother's nails, as well as her legs, are quite characteristic of eczema. My belief is that this transmutation is common in the hereditary transmission of morbid tendencies. I believe that it occurs very frequently between psoriasis and chronic eczema, and not uncommonly between ichthyosis and both of these. What is

inherited is probably some peculiarity in the structural endowments of the skin. To this is added, during the patient's lifetime, the influence of some local exciting cause, and a form of dermatitis is set up which proves infective in the patient's tissues. If it begins as eczema it will keep to eczema, if as psoriasis to psoriasis. You may, however, in some instances, have mixed cases, and forms of dermatitis in which it is doubtful whether they should be called psoriasis or eczema are not very uncommon. These doctrines are, I submit, of very considerable importance in enabling us to realize the true nature of the maladies in question. The doctrine of transmutation in hereditary transmission finds illustrations in many departments besides skin diseases, and notably in that of new growths.

Herpetiform Morphœa.

The subject of this case was a well-grown girl, aged 11, named Coleman, of whom several drawings and a wax model have been taken at different stages of her malady. She is the subject of ivory-patch morphœa, arranged in zones on the shoulders, trunk, and upper parts of the thighs. The disease has now been in existence for several years, and the ivory patches are becoming much thinner. They still, however, admit of easy demonstration. No new patches have been produced, nor have the old ones extended at their edges.

Comments. I will not say more about this case at the present time than that it is one of the best examples that I have seen of what may be called "Morphœa in zones." It is the patient's trunk only which is involved, and the ivory patches slope downwards and forwards in belts, with intervals of healthy skin, but with almost exact bilateral symmetry. You might at first sight be disposed to say that the disease was symmetrical; but although it is extensively bilateral, it is not exactly so. Thus there are on each side certain patches which have no representatives on the other. This is important as corroborating the view that the disease is located by nerves. The adjective "herpetiform" is used in order to denote the fact that the patches are distributed by nerves just as zoster is. It is true that zoster is very rarely indeed bilateral, whilst morphœa is often so. This difference, however, probably only implies that the initiating influence in morphœa is placed more centrally, that is, in the spinal cord itself, and thus affects both sides at once. A moment's thought will convince you that the distribution of any malady in zones or bands, although these may be bilateral, must imply nerve-causation.

THERAPEUTICAL NOTES AND FORMULÆ.

Wounds.—*Compound Tincture of Benzoin as a Surgical Dressing.*—Carefully cleanse wound, removing all foreign substances; irrigate with antiseptic solution if there is any evidence of infection. Thoroughly check all hæmorrhage, and place a layer of surgical cotton around the wound, pouring the compound tincture of benzoin next to the surface of the latter, and saturating the cotton immediately surrounding the injured tissues. This drug, after undergoing evaporation, will form a coating with the cotton that will hermetically seal the part, thereby rendering it perfectly aseptic. The dressing can be left intact for six or seven days; if it become loosened, the patient may add a little more of the tincture.

(*American Therapist.*)

Football and Albumenuria.—Dr. Macfarlane has examined the urine of thirty-five presumably healthy men after playing football, and found in every case indications of the presence of albumen and also casts (epithelial hyaline and blood.) He briefly discusses the meaning of the condition, and concludes that much fuller statistics are required before any just conclusions can be drawn as to any possible harm resulting.—(*Med. Rec.*)

For Rheumatism.—The following prescription is recommended by the *Journal de Medecine de Paris* :—

R   Salol   ...   .
    Etheris   ...   ...   aa   p. iv
    Collodii ...   .   p. xxx
M.   Sig.: To be painted about the painful and inflamed joint.

Hemicrania.—The following formula is recommended in the treatment of migraine :—

R   Citrate caffeine   ...   gr.xv
    Phenacetine   ...   ...   gr.xxx
    White sugar   ...   ...   gr.xv
Sufficient for 10 capsules. Sig.: One every three to four hours during the period of the attack.

(*Gazet. Med. di Roma.*)

A CLINICAL LECTURE

ON

BENIGN GROWTHS IN THE LARYNX.

Delivered at St. Thomas's Hospital, January 15, 1895, by
FELIX SEMON, M.D., F.R.O.P.,

Physician for Diseases of the Throat to the Hospital.

GENTLEMEN,—The chapter on benign growths in the larynx may truly be said to date its origin from the introduction of the laryngoscope. The proof of this statement will be found in the fact that, whereas previous to the introduction of the laryngoscope the total number of benign growths in the larynx observed and reported in the whole history of medicine amounted to about sixty altogether—and most of these were not discovered until the post-mortem examination—hardly thirty years later, in 1888, I was able to bring together in the course of a collective investigation, to which I shall afterwards have to draw your attention, not less than 10,747 cases observed by 107 laryngologists between 1862 and 1888. Needless to say, even this number does not nearly represent the total number of cases of benign laryngeal growths which have actually occurred during that time, but the number adduced will not merely justify my introductory statement, but at the same time show you that laryngeal growths are not nearly so rare as at one time they were believed to be. As chance will have it, quite a number of cases in point are at this moment under treatment in our department, so that you have the opportunity of practically studying the subject.

With regard to the causation of benign growths of the larynx the same obscurity obtains which reigns with regard to the causation of new growths in general. It is customary to look upon chronic laryngeal catarrh as the most prolific source of the formation of innocent laryngeal tumours. I venture to opine, however, that this belief is one which urgently needs revision. I am not aware that a case has ever been described by a trustworthy observer in which, during the progress of

a laryngeal catarrh or inflammation, one of the more common forms of new growths actually made its appearance under the eyes of the observer; whilst the existence of congenital new growths in the larynx, and the fact that in the great majority of innocent growths observed at other times of life, there is no evidence of any inflammatory process in their neighbourhood, certainly points in the direction that chronic inflammation is, if a possible, yet certainly not an indispensable factor in the production of these formations. That occasionally catarrhal processes exist in association with new growths in the larynx is undeniable; it is, however, quite open to question whether in these cases the catarrh is not the consequence rather than the cause of the existence of the new growth.

With regard to the time of life at which innocent new growths of the larynx may make their appearance, it may be stated that no age gives immunity against their formation. In the museum of the Great Ormond Street Hospital for Children you may see a specimen of the larynx of a child which died about thirty-six hours after birth from suffocation caused by a large cyst in the larynx. This case has been described by the late Dr. Arthur Edis. Often enough also in the cases of multiple papillomata in the larynx of small children, some of which we have every year the opportunity of observing in the hospital, you will hear the statement that the child's voice had been hoarse from birth. On the other hand, I have myself removed an enormous cyst, which threatened suffocation, from the larynx of an old lady aged over 80, the history of aphonia and dyspnœa only dating back a comparatively short time; and other cases of innocent growths at extreme old age have been described, the history of which pointed to a comparatively recent origin of the new formation.

Whilst thus no age is quite immune, there can be no doubt that two periods of life are most prone to the disease. In the first place, it is the age from 20 to 40 in which benign growths are most frequently met with; secondly, the very first years of life. With regard to the first class of cases, let me at once tell you a good rule. If you find a new growth in a person over 50, in which

the history and symptoms do not point to the disease having existed for a long time, you ought to look upon it with suspicion, however innocent it may look, as experience teaches that growths originating at that time of life are much more frequently malignant than benign. I shall have to return to this subject later on.

There can be no doubt that men are more frequently attacked than women. The difference between the two sexes, however, is not such a striking one as in the case of malignant growths of the larynx, in which the male sex shows such a preponderance that the proportion is at least three to one. Whether occupation exercises a marked influence is also a question still open to discussion. It is usually assumed that professional use of the voice predisposes, in a marked degree, to the formation of new growths, but in a comparatively large number of my own cases no such influence could be traced, with the exception of the small circumscribed thickenings of the vocal cords mainly observed in the cases of singers, who either overuse or do not know how to properly use their vocal organs; and I think that this question of occupation is another one in which a revision of facts would be desirable.

With regard to the various forms of benign growths in the larynx, they are of exceedingly various histological structure. Thus we meet with papillomata, fibromata, cysts, lipomata, myxomata, angiomata, ecchondromata, adenomata, lymphomata, and quite recently several cases have been described in which intra-laryngeal growths have been found to consist of normal thyroid tissue, and to owe their origin, no doubt, to hyperplasia of aberrant particles of the thyroid gland.

For practical purposes of all these classes of new growths three only need be considered—viz., papillomata, fibromata, and cysts. All the others named are so very rare that they may be looked upon in the light of pathological curiosities. Matters are very different with regard to the first-named classes. Of these papillomata are by far the most frequent. In the collective investigation which I mentioned at the commencement of this lecture, it was ascertained that in fully 39 per cent. of all benign growths of the larynx the histological structure of the new growth was of the nature of papilloma. Next in frequency comes fibroma, and then, after a long interval, cysts.

Laryngeal papilloma occurs either in a solitary or a multiple form. It is met with at all periods of life, and particularly in children it is almost the only form of benign laryngeal growth (except a few rare cases of fibromata and cysts) that comes under observation. The vocal cords are its favourite seat, and of these again the anterior commissure of the cords and the anterior thirds. Papillomata of the ventricular bands come next in frequency, and are generally met with in cases of multiple papillomatous degeneration. In some cases the papillomatous growth is seen to emerge from the ventricle of Morgagni. Papilloma of the epiglottis is decidedly rare, and in persons above 50 should always be looked upon with suspicion as being probably malignant. The same obtains with regard to papillomatous-looking growths on the arytæno-epiglottidean fold, and on the arytænoid cartilages. In a recent case described by Mr. Shattock and myself, which has given rise to a great deal of discussion and controversy, it was the situation on the arytæno-epiglottidean fold of a growth that at first, under the microscope, seemed to show the characters of genuine papilloma, which at once attracted the suspicion that its nature might be otherwise than benign—a suspicion which was justified by the further course of the case. Never, so far as my knowledge goes, will you find a genuine papilloma in the inter-arytænoid fold—not even in cases of almost general papillomatous degeneration of the laryngeal mucous membrane; and I may tell you here at once that this locality, according to the observations and descriptions of the most experienced laryngologists, appears to have a sort of immunity against the occurrence of benign growths. Only one authenticated case has recently been reported by an Austrian observer, and this case was reported so shortly after the removal of the new growth that it will be necessary to wait for the further development of events before definitely admitting its innocent nature. I have brought here quite a number of specimens, showing solitary as well as multiple papillomata of the larynx, and you will see that even in the most extensive cases the inter-arytænoid fold has remained perfectly free. If you should meet with something which at first sight looks like a genuine papilloma in the inter-arytænoid fold, you will always have to think of the so-called "false excrescences" of syphilis and tubercle, and in many cases you will find that the apparent excrescence represents merely the steep edge of an ulcer on the posterior wall of the larynx seen in profile.

Laryngeal papillomata consist of a number of papillæ containing vessels and covered by an epithelial layer. They are almost always more or less pedunculated, resemble bunches of cauliflower *en miniature*, and are of white or pinkish colour. If in an adult of over 40 years a papilloma be met with which is of a particularly snow-white colour, and the individual protuberances of which are not rounded, like those of ordinary cauliflower, but of a more pointed nature, so that the appearance of the whole growth would remind one rather of a meadow covered by hoarfrost than the usual cauliflower form, my experience has taught me to look upon such apparent papillomata with suspicion, as usually they represent merely papillomatous excrescences from an epitheliomatous basis. Ordinary papillomata, when removed, show a marked tendency to recurrence, particularly multiple ones. Their removal is not attended, as taught even now in a great many text-books, by any considerable hæmorrhage, as you have repeatedly, during this term, had the opportunity of convincing yourselves.

Now we come to the fibromata. Histologically they consist of connective tissue with admixture of elastic fibres; they are covered by epithelium, and are more or less richly supplied with bloodvessels. In my own experience the vascularity, particularly that of the sessile forms, is very considerable, and the hæmorrhage on removal often is much greater than in the case of papillomata. Fibromata are met with in the larynx in very different sizes, from the small nodules which, particularly in singers, are not rarely met with symmetrically on the border of the anterior and middle thirds of the vocal cords, and which are not larger than a pin's head, to truly enormous growths filling the whole larynx, of which I show you an example here. They occur, clinically speaking, in two forms, the sessile and the pedunculated, and in both these forms the growth is almost always solitary, two fibromata having been very rarely simultaneously observed in the larynx, whilst multiplicity, such as is common in papillomatous disease, does not exist for these forms. Sessile fibromata are almost always semi-globular, sometimes slightly mammillated, of a white, pink, red, or even bluish colour, and their most favourite seat, as indeed of all benign laryngeal growths, are the vocal cords. In the pedunculated form the stalk may be small or broad, long or short. Sometimes it is long enough for the growth to hang down into the subglottic cavity, and to become visible only on forced expiration, or cough, when it may be thrown above the level of the vocal cords, whilst on deep inspiration it is sucked into and may completely disappear in the subglottic cavity, the vocal cords on the next phonation meeting over it, and only a slight inequality on one cord, which indicates the origin of the pedicle, betraying its existence. The pedunculated fibromata also greatly vary in size, from a pea to a hazel nut or more.

Laryngeal cysts are almost always of glandular origin, and are of the nature of retention cysts. They may occur in any part of the larynx in which glands pre-exist, but are most frequently met with on the dorsal aspect of the epiglottis. Sometimes they attain such a large size as to be visible with the naked eye when situated in that locality, whilst if arising in the larynx itself they may actually threaten suffocation.

Of the other forms of laryngeal growths I have already told you that they may be looked upon as mere pathological curiosities; and I will only show you here two illustrating specimens. The first is a very rare tumour indeed, an enormous lipoma, which was removed by our former senior surgeon, Mr. Sydney Jones, from the right arytæno-epiglottidean fold. Part of the growth actually projected into the man's mouth, which you can easily understand when looking at its enormous size. The second one is a specimen of another rare form of growth, of which there are only five or six cases on record. It is a myxoma which I removed some time ago from a woman, aged 26, who had been suffering from hoarseness almost amounting to aphonia ever since her birth. It was situated in the anterior commissure of the vocal cords, and the voice became normal immediately after its removal.

Having thus described the various forms of benign growths met with in the larynx, we next come to the consideration of the symptoms which they cause. Needless to say, they will greatly vary according to the size and to the situation of the growth. By far the most frequent symptom met with, and indeed in most cases the only one, is more or less vocal disturbance. This explains itself when you remember that the vocal cords are the predilection seat of all benign growths. The degree of vocal disturbance will, of course, vary according to the degree of the interference with the free vibrations of the vocal cords; from slight

hoarseness to complete aphonia all intermediate stages are met with, and it is a remarkable thing that even small growths, when situated in the anterior commissure of the vocal cords, as a rule produce much greater vocal disturbance than larger ones originating from the middle parts of the vocal cords. Should the growth, of whatever histological nature it may be, attain such a size as to considerably narrow the canal of the larynx, dyspnœa will be the natural result, and the degree of this dyspnœa will again depend upon the amount of narrowing caused. Pain is practically never caused by benign growths in the larynx, and only in rare cases, particularly of pedunculated growths, do patients speak of strange sensations in the throat. Cough also is a very unusual symptom; dysphagia will only be produced when a large growth is situated on the dorsal aspect of the epiglottis, and spontaneous hæmorrhages from benign growths are practically unknown.

I have now to speak of the possible developments which these growths may take. It has already been mentioned that papillomata occur either in the solitary or the multiple form. In the former case they may for a long time remain stationary after having attained a certain size, but are more likely to gradually become larger, and this is, indeed, the rule with the multiple forms. I show you here an almost incredible quantity of papillomata which I removed in 1884 from the larynx of a lady aged about 45; they do not represent recurrences, but were all simultaneously present, and, needless to say, caused the highest degree of difficulty of breathing. The case possesses a certain additional interest, inasmuch as it was the first, so far as I know, in which in this country cocaine was utilised for intra-laryngeal operations.*

Fibromata, after having attained a certain size, not rarely become stationary; in other cases, however, they continue to grow slowly, and may sometimes, after many years, cause serious respiratory difficulties.

In extremely rare cases a spontaneous expulsion of new growths has been reported. This, however, is an event of such excessive rarity that it ought not to be held out as a chance to any patient. In a few cases of growing children with small apparently fibromatous nodules on their vocal cords, I have myself seen involution take place in the course of years, but this too is certainly very rare.

* "Lancet," vol. ii., 1884, p. 912.

On the whole it may be said that benign laryngeal growths, when left to themselves, though they may become stationary at a certain period, are more likely to gradually increase in size. Papillomata do so sometimes rather rapidly.

Here I wish briefly to touch upon a question which of late years has caused a good deal of controversy, viz., whether benign laryngeal growths ever undergo malignant degeneration, and, if so, whether this tendency is increased by intra-laryngeal instrumental interference. Assertions answering this question in the affirmative have been periodically made in the course of the last seventeen or eighteen years, and reached their culminating point at the time of the illness of the late Emperor of Germany. As their correctness or incorrectness could obviously only be tested, and the question be definitely settled, by the experience, not of a single observer, but of the great majority of those who had had special opportunities of observing laryngeal growths, I instituted the Collective Investigation repeatedly mentioned in the course of this lecture. Its result was the following:—Of 10,747 cases of innocent laryngeal growths observed by 107 laryngologists, 8216 had been intra-laryngeally operated; of these in 33 cases malignant degeneration was reported, that is to say, 1 degeneration in 249 cases; but on critically analysing the individual cases of reported degeneration, in 5 only was such degeneration found to have been quite, or almost, undeniable, so that the proportion, if these only were admitted, would be 1 degeneration in 1645 cases of operations. If 7 further cases, in which the degeneration was more or less probable, be added to the number of the certain cases, the proportion of degeneration would be 1 in 685 cases. The remaining cases of reported degeneration were of a most doubtful character, and in most of them it was probable that a diagnostic mistake had been committed from the very beginning. Under all circumstances, therefore, the occurrence of a malignant degeneration of a previously benign growth must be considered as an event of the greatest rarity, and the very number I have adduced will show you that the fears which were raised as to the influence of intra-laryngeal instrumentation upon the occurrence of such a degeneration were absolutely unfounded. The best proof of this fact is, that actually more spontaneous degenerations in non-operated cases were reported in the Collective Investigation than degenerations after removal, the percentage in the

first class of cases being 1 to 211, whilst in the second class it was 1 to 249. That theoretically a possibility exists of the degeneration of a benign growth into a malignant one, no reasonable observer will deny. There are plenty of instances in other parts of the body, and in tissues of the most varied histological character, in which their occurrence is a matter beyond doubt (I need only remind you of the fact that the hyperplastically changed thyroid gland, *i.e.*, goitre, is more prone to malignant disease than the normal one). But that a "special" tendency should exist of benign laryngeal growths to undergo malignant degeneration, that this occurrence should be frequent, and that it should be favoured by intra-laryngeal instrumentation, may now be looked upon as an absolutely exploded theory.

We next come to the question of diagnosis. Needless to say, the diagnosis of a benign growth in the larynx can only be made by means of the laryngoscope. Its usual symptoms—hoarseness, and, in some cases, dyspnœa—are much too ambiguous to allow of any other means of diagnosis than direct inspection of the affected part. They may belong as well to inflammatory, syphilitic, tubercular, paralytic, or malignant disease of the larynx as to a benign growth; and, although in the rather rare cases of large pedunculated fibromata, which may be sometimes thrown above, sometimes be impacted between, and sometimes be sucked below the vocal cords, a very experienced observer may form, from the suddenly varying character of the voice, a certain suspicion as to what he would be likely to find, this suspicion ought not to be trusted to under any circumstances, and the only way, I repeat, to ascertain what the cause of the hoarseness and dyspnœa in such cases may be, consists in the use of the laryngoscope.

The differential diagnosis between benign growths in the larynx on the one hand, and the diseases just named, is, on the whole, not difficult. By means of the laryngoscope you will, in the great majority of cases, easily enough distinguish between a growth and a catarrhal, syphilitic, or tubercular swelling of a part of the larynx, whilst in the cases of a paralytic hoarseness, or the impaction of a foreign body, etc., the differential diagnosis can never offer any difficulty. More difficult, and, indeed, often impossible, is the differential diagnosis between benign and tubercular tumours. In the course of last term we had a

patient here in whose case I removed a growth from the anterior commissure of the vocal cords, in the belief that it was either of a fibromatous or of a malignant nature. I was not a little surprised when the microscopic examination made by Mr. Shattock conclusively demonstrated that it consisted of mere *débris*, containing numerous giant cells, and when the bacteriological examination showed the presence of tubercle bacilli. Fortunately these tubercular tumours are so extremely rare that it is not very likely you will ever come across one of them.

Very difficult in many cases is the differential diagnosis between benign and malignant growths in the larynx, particularly in the early stages of the latter. A growth may look—and I wish to strongly emphasize this point—absolutely like an innocent one, and yet, *from its very commencement*, be malignant. The main points which ought to raise your suspicion as to possible malignancy in the case of an apparently innocent laryngeal growth are the following:—

1. The patient's age. Although innocent growths, as already mentioned, occur in the larynx at any period of life, they are distinctly rare after 50, and not very frequent between 40 and 50, whilst malignant growths, in the great majority of cases, occur between 40 and 70. Thus, it is particularly the decade between 40 and 50 which is common to both classes of growths, in which the greatest circumspection is necessary, whilst, if in a patient of over 50 afflicted with a laryngeal growth, the history points to a short existence of the new formation only, the probability is greater that it will turn out to be malignant.

2. The situation of the growth. Benign growths of the larynx—unless there is a general degeneration of the laryngeal mucous membrane—show a tendency to be localized in the *anterior* parts of the vocal cords. The occurrence of a growth on the posterior parts of the cords, on the arytæno-epiglottidean fold, on the epiglottis, on the arytænoid cartilages, particularly if this should happen in an adult over 40, adds a further element of suspicion.

3. Benign growths may, from their size, mechanically interfere with the free movements of the vocal cords, but they will not intrinsically hamper the movements of the cord to which they are attached. In malignant growths, on the other hand, sometimes even at an early period, a sluggishness in the movements of the cord to

which the new formation is attached is observable, due, no doubt, to the infiltrating character of the malignant disease. Later on this sluggishness may increase up to complete immobility of the affected cord. This I have found to be a very important symptom ; it is, however, not invariably present, inasmuch as also in malignant growths the original infiltration may be of a more superficial character.

4. If a well-defined growth in the larynx of an adult be surrounded by a limited area of well-marked inflammation, this again points in the direction of malignancy. (It may be observed here that the existence of this area of congestion is sometimes an element of difficulty in the differential diagnosis between Virchow's pachydermia verrucosa and malignant tumours of the larynx when situated near the vocal processes).

5. If in an adult one vocal cord seems to be entirely involved in a papillomatous fringe extending from the anterior commissure to the vocal process of that cord, whilst the opposite side is perfectly normal, this again is a very suspicious moment, pointing to malignant disease in papillomatous form of the ventricle of Morgagni.

6. I have already mentioned that unusual snow-white colour and grass-like appearance of a papilloma point to malignancy.

Such, gentlemen, are the points which on laryngoscopic examination ought to attract your attention from the point of view of differential diagnosis. They do not exhaust the possibilities which may here come into question, but are those which, in the present state of our knowledge, I have myself found most serviceable in making a differential diagnosis. If you can intra-laryngeally remove a portion of a suspicious growth, and if this, on microscopic examination, shows the character of squamous-celled carcinoma (epithelioma), of course your doubts are solved, but, remember, that if the result of the microscopic examination be doubtful or even negative, you are not entitled to cast to the winds your well-founded clinical doubts, and to throw the responsibility which belongs to the clinical observer upon the microscopist. More on this point I shall have to say when we discuss malignant diseases of the larynx. In conclusion of this question of differential diagnosis, I hope that in cases of rational doubt as to the nature of a growth you may have observed in the larynx, you will do your best to arrive at a definite opinion as soon as possible,

Should, after all, the growth turn out to be malignant, the sooner a radical operation is undertaken, the better are the patient's chances.

The prognosis of benign growths of the larynx nowadays is universally good. It will hardly ever happen, as it did in former days, that a patient is simply choked by a very large new growth, and in most cases intra-laryngeal removal of the new growth will free the patient from his disease, although the tendency to recurrence of papillomata must always be taken into consideration. Also the prognosis with regard to recovery of voice is, on the whole, very good, though in cases of sessile or very multiple growths some small vocal disturbance may remain behind after their removal. The one class of benign new growths in which the prognosis, if not to life yet at any rate to duration of disease and to subsequent function of the parts, ought to be very guarded, are the cases of papillomata in early childhood. Although in a few of these cases intra-laryngeal operation even at a very early age appears to have been brilliantly successful, yet in the much larger number it has been found necessary to either perform prophylactic tracheotomy in order to prevent suffocation, or to proceed to thyrotomy and thorough removal of the new growth, which, however, I am bound to say, has often enough not been successful, inasmuch as after a short time recurrence of the papillomata took place. Even in these cases, however, I think that when once a general consensus of opinion has been obtained as to the best manner of proceeding, an ultimate good result can be hoped for. With this I come to the last part of my lecture : the question of treatment.

On this point I shall be brief, although the treatment of benign growths in the larynx forms one of the most brilliant achievements of medical science in the course of the last thirty years. But this treatment demands a *technique* which no amount of theoretical instruction will ever impart, and in order to be successful must be studied by patient and long-continued practice, without which its adoption is certainly not free from risks. I shall therefore be content to give you a few general principles upon which to act, and for the rest only briefly summarize the present modes of dealing with intra-laryngeal benign growths.

In a few cases of these growths no treatment whatever may be either required or indeed advisable; thus occasionally small fibromata are met with on the vocal cords, which cause such slight

symptoms as to be hardly appreciable. In these cases removal sometimes is exceedingly difficult, and the risk of injuring healthy parts in the neighbourhood great enough to contra-indicate operative removal, unless it be urgently desired by the patient himself, and unless the operator possesses full mastery of his *technique*. Such cases, however, form the exception, and in the great majority it will be desirable and even necessary, on account of the symptoms caused by them, to remove the neoplasms. Although I am very well aware that statements have been made to the effect that by astringent local treatment some growths have been made to disappear, all I can say is that I have never met with a well-defined neoplasm of the larynx which has yielded to such gentle treatment, and I consider its employment a mere loss of time. The only efficacious method I know of to get rid of benign neoplasms of the larynx consists in their removal by operation. The long and bitter contest which has been waged as to the relative advantages of intra-laryngeal and extra-laryngeal removal between laryngologists and general surgeons has now been practically definitely decided in favour of the intra-laryngeal method, and the number of instances in which external operation has to be resorted to in these cases is becoming more and more limited, and is at present practically restricted to sessile intra-laryngeal growths situated in the subglottic cavity or even further down. The introduction of cocaine for intra-laryngeal operations has been simply invaluable, and has no doubt much contributed towards this consummation ; but even prior to its employment the general preferability of the intra-laryngeal method had been almost universally acknowledged, and the principle to be followed can briefly be summarized by the dictum : that an external operation in a case of a benign growth of the larynx is only indicated when an experienced laryngologist has failed to remove the neoplasm *per vias naturales*. As to what method ought to be employed, *i.e.*, whether forceps, the snare, the galvano-cautery, protected knives, etc., ought to be used, cannot be theoretically taught, and will in every individual case depend upon its particular features, and also to some extent upon the habits of the operator. A very excellent instrument recently introduced and suitable for many forms of growths situated on the vocal cords, though not for all, is Dr. Dundas Grant's safety-forceps. Often more than one form of instrument may have to be used in the

same case, and from general experience I think I may say that even now intra-laryngeal operations are not the easy thing which they were represented to be in the first enthusiasm after the introduction of cocaine. Some cases now, as before, baffle the skill and the patience of the most experienced operator for a long time, and recurrences of papilloma are now as frequent as ever. With sufficient perseverance, however, on the part of both patient and operator, ultimately a very satisfactory result may be confidently anticipated in the overwhelming majority of all cases.

A few words are necessary, in conclusion, concerning the treatment of multiple papillomata in children. These cases have been, and still are, the greatest crux with regard to both diagnosis and operation. With regard to the former it is, as a rule, extremely difficult to make a laryngoscopic examination in small children, though there are very surprising exceptions to that rule, and the employment of a general anæsthetic has been found to be of little avail, because the salivation ensuing under the use of that method and covering the reflecting surface of the mirror renders satisfactory examination in most cases quite impossible. As to operation the intra-laryngeal method has succeeded in very few of these cases only, and thyrotomy has, as already mentioned, given no immunity against the recurrence in a great many instances, in spite of apparently very thorough removal of the new growths. The plan which I have so far followed consists in performing tracheotomy, if unavoidable, and postponing removal of the growths themselves to a later period of life, when the child is reasonable enough to allow of intra-laryngeal interference. Intubation, which has been recommended under these circumstances with a view of both doing away with the dyspnœa and causing absorption of the new growths, appears to me a proceeding the risks of which hardly justify its adoption; it stands to reason that soft multiple papillomata are very apt to be torn away by the introduction of the tube, and to be pushed into the lower air passages, where they may set up dangerous mischief. Moreover, I do not know of any authenticated case in which real absorption of the growths has been obtained by this method, and I can easily conceive that if the intubation tube should become dislodged from its position by some accident, whilst no skilled assistance was near, the dyspnœa suddenly arising from the rush of blood into

the previously compressed growths may be of the most serious nature. On the other hand, it is to be earnestly hoped that the method recently introduced by Dr. Scanes Spicer may turn out to be generally useful. It consists in combining general chloroform narcosis with frequently repeated local mopping of the pharynx and larynx of the patient until all secretion is thereby arrested, when, according to the author's experience, it is possible to laryngospically examine the patient, and, if necessary, to at once proceed with the removal of the new growths, should such be found. The method is at present still a somewhat complicated one, demanding a good deal of assistance, and further experience is required before an ultimate judgment as to its general utility can be given. Let us hope, however, in the interest of many small patients, otherwise condemned to wearing of the tube for years or to the repeated performance of thyrotomy, that the expectations which the inventor bases upon his discovery may be fully realized.

THE TREATMENT OF NEURITIS AND SEVERE NEURALGIAS.

A Paper read before the Islington Medical Society on Tuesday, Jan. 22, 1895, by

JAMES CAGNEY, M.A., M.D., M.R.C.P.,

Physician to the Hospital for Epilepsy and Paralysis; in charge of the Electro-Therapeutical Department, St. Mary's Hospital.

GENTLEMEN,—The attempt to deal at one time with the conditions which are combined in the title of this paper will, I think, be justified by a little reflection. The term neuralgia is a vague one. I question if anybody has a clear view of what he means when he uses it. It certainly means different things to different people. It belongs to a class of provisional epithets of which a good many still survive in medicine—destined to be shorn gradually of their significance and finally, it may be hoped, to drop out of use entirely.

The word neuralgia at one time meant pain of almost every kind for which an obvious cause was not at once discernible. With the advance of knowledge and the acquisition of facilities for observing causes became obvious which before were

not suspected, and the denotation of the term neuralgia lessened. There is at present a possibility, a necessity to diminish it still further, and this chiefly by making closer and more general the study of peripheral neuritis. I believe that most cases of neuralgia are neuritis. That is why I propose to deal with the two affections together. It is necessary to determine the relation of the one to the other. If this be not done neither can be treated of satisfactorily, because there is a great danger that they will be mistaken for each other.

When a physician uses the word neuralgia he perhaps intends chiefly to enforce a negation; he means that the pain of which he speaks is not due to any one of a number of conditions of which he knows something. Besides this, however, there are certain conceptions attaching to the idea of neuralgia. It is necessary briefly to review some of these, and decide if we can how far any of them may usefully serve as marks by which the condition may be distinguished from others.

1, Neuralgia is a pain usually conforming in situation to the distribution of peripheral nerves. This mark belongs essentially to neuritis.

2. It is conceived to be independent of any gross lesion. Here very much depends upon the skill of the observer—very much upon the state of knowledge. Thus there was a time when gastric ulcer would generally be dealt with as gastralgia in particular cases, and must often be so mistaken now. A distinction of this kind is useful only in rare and very happy combinations of circumstances.

3. The presence of what is known as "tender points" is sometimes thought to be a mark of neuralgia.

Now, I think I have never seen a case of extensive neuritis in which such tender points were absent. Further, they are usually placed upon bony prominences, or where the nerves pierce the fasciæ and aponeuroses—where, in fact, the nerves are subject to injury or pressure, and if in such places there is tenderness, it would seem that it is the nerve which is tender, and therefore, in the absence of evidence to the contrary, inflamed. This question of evidence is nearly always open, and where it can be settled, the solution is very much in favour of neuritis. There is under my care at present a man who came to hospital complaining of occipital neuralgia, with tender points in the distribution of the great occipital nerve. He was not aware of being otherwise ill.

I found his urine of high specific gravity, and containing abundance of sugar. The neuralgia began on a certain day three weeks before. On that day also he fainted, and he had just begun to suffer from thirst, and to pass great quantities of water. Did the pain depend on neuritis? It is a question of probability. With the knee-jerks present, and no motor implication, there can be no certainty, but the tender points will not settle the matter. On the one hand, diabetes is a potent cause of peripheral neuritis; on the other, I would urge the importance of remembering that the action of other poisons which select the peripheral nerves, as alcohol, lead, and the toxic agent in gout, are apt to have at the same time a specific effect on the central nervous system. It will be urged presently that a central sensory irritability is the one condition to which the term neuralgia may usefully be applied, and one is led by analogy to admit the possibility of a diabetic neuralgia as well as of a diabetic neuritis. A case such as this is generally cleared up by later developments.

4. Neuralgic pains are said to be spasmodic. Toothache, to take a familiar instance, is apt to be spasmodic. The inflamed pulp can determine pains outside itself. We must distinguish between the pain in the pulp and the rest of the pain. The latter seems to imply a central agency, and if the spasmodic character depends upon that it is a useful distinction. Into this I will not enter, but it suggests matter to think about.

5. Periodicity is a characteristic of some forms of so-called neuralgia—notably of that belonging to malaria. I will not dwell on the fact that malaria is a recognised cause of peripheral neuritis, but I mention a case which taught me to remember that periodicity attaches to other pains besides those of ague. I had care of a woman some 40 years of age. She was sent to me to be treated for neuralgia of the head and arms. This was described as being of most intolerable severity. It occurred at approximately regular hours twice in the day. The patient had resided long in a malarial district, had had typhoid fever while there, and had a large spleen. A constant current subdued the pain, but neither quinine nor any other drug was effectual. Careful investigation showed a reaction of degeneration in some muscles of the upper extremity. It was therefore a neuritis, but the distribution of symptoms was not that of malarial neuritis. The possibility of syphilitic infection

had been dismissed on better authority than mine. Nevertheless, in course of time a most undoubtedly syphilitic skin lesion developed, and in a short time the case was cured by large doses of iodides. This was nearly the best instance of periodicity that I have met with, and it occurred in a case of syphilitic neuritis.

6. When all the kinds of pain have been investigated, pathologically and otherwise, there must remain some which can reasonably be referred to an excessive irritability of the sensory centres, and for such pain the term neuralgia will probably be reserved. It is possible that this irritability is spasmodic, just as the effect of central motor irritation is spasmodic. It is possible also that certain causes—poisons which obey a law of periodicity—induce this condition. I content myself with alluding to Laveran's researches in malaria as grounds for detaching the notion of periodicity from any central cause whatever.

If neuralgia is vague, neuritis is precise enough. It is a condition familiar both pathologically and clinically. It is easily recognisable, and in most cases with the utmost certainty. For all that, it is likely that in a large proportion of instances it is overlooked. Two varieties are distinguished.

Interstitial neuritis is the name given to a condition in which the nerve sheaths are inflamed. It might more aptly be called neuro-cellulitis. I don't know whether it ever has been so called. The other form, *parenchymatous neuritis*, is an affection of the nerves themselves. It is not an inflammation; there are no vessels in a nerve at any time, and the classical manifestations of inflammation are wanting. What occurs is essentially the same as that which takes place after section or destructive injury of a nerve. There it is called degeneration, here it is neuritis, which should mean nerve inflammation. Moreover, interstitial neuritis, when bad enough to produce symptoms, is parenchymatous neuritis plus inflammatory changes in the sheaths. Altogether the terminology is not happy, and I shall have occasion to urge the need of not being misled to regard the essential lesion as an inflammatory one.

This lesion, however, is attended by symptoms which there are no mistaking. In the motor system they are wasting, weakness, flabbiness, and usually great tenderness of muscles; but above all a varying degree of the reaction of degeneration. On the sensory side there are pain, tenderness, and the different forms of anæs-

thesia in the distribution of particular nerves, which if accessible may be found to be enlarged. Trophic changes are herpetic and other eruptions, ridged nails, glossy skin, joint affections, errors of secretion, and other changes. There may further be psychical affections dependent upon the same cause, as alternating delirium and coma in alcoholic neuritis; irritability and melancholia in gout; hysteria in lead poisoning; stupor and dulness in toxæmia. My colleague Dr. Guthrie has done good service in showing the tendency of the diphtheritic poison to work suddenly and fatally upon the vital centres in the medulla. So many and so precise are the signs of peripheral neuritis, that with reasonable care it can rarely be overlooked. If by neuralgia be understood pain due to a central irritability, while all other pains are thought to be peripheral, these latter will be extrinsic and intrinsic. The intrinsic peripheral pains are the pains of neuritis. I suggest this classification as one which may be helpful in dealing with the important subject of treatment. It must be borne in mind, however, that a central pain and an intrinsic peripheral pain may, and very commonly do, exist together.

Treatment. The classification briefly made here notices pain which is extrinsic to the nervous system. This notice is necessary, since all pain has to do in some way with the nerve structures. Pain which is extrinsic in the sense intended is foreign to the present subject. Such is the pain of a wound or that caused by a cancerous growth. It is important, however, to be able to exclude it in our notions of causation. We often speak or hear spoken of "referred pain." Thus a gentleman is now under my care who, some months ago, underwent an operation for hæmorrhoids. He shortly after began to suffer from pain in the distribution of one sciatic nerve, and this, he was told, was "referred." The condition was indistinguishable on other grounds from an acute sciatica, and there is foot-drop, muscular wasting, and reaction of degeneration. The patient is gouty and has what is known as a "torpid liver." He is the subject of hæmorrhoids and of a sciatic neuritis, dependent upon a common cause, but in this case the term "referred pain" is clearly misapplied. Another case will illustrate the subject better. The fifth nerve, it is well known, is peculiarly apt to be the seat of pain. One reason for this may be the great number and distribution of delicate and vulnerable terminal organs which

belong to it. Some of these appertain to the teeth, others to the eye, and others, again, are distributed on mucous surfaces. A woman came to the hospital complaining and showing evidence of intense paroxysmal pain in the distribution of the infraorbital nerve of the left side. This was attributed to a kick, and had lasted twelve years. A breath of cold air, the effort to speak, mastication, and almost any movement, brought on a paroxysm. Her sleep was broken, and in her waking hours for twelve years she was seldom for half-an-hour without excruciating agony. Every drug had been tried; all her teeth had been drawn, and every attempt to relieve her had been ineffectual. She was anxious to have an operation performed; but I found that the application of a constant current eased the pain. Subsequent investigation showed that there was necrosis of the middle turbinated bone, and removal of the diseased tissue effected an immediate cure. The anatomical relations warrant the use of the term "referred pain" here, and as a practical suggestion I would counsel the examination of the nose in trigeminal neuralgia before the extraction of sound teeth. In analogous instances it is expedient to search for sources of irritation with a view to their removal.

Restricting our attention now to the treatment of pain, which is intrinsic to the nerve structures, it may be premised that such treatment may be general or local, and the remedies, if drugs, are spoken of as anodynes. Almost any drug may be an anodyne, and so it is usual to speak of anodynes as indirect and direct. Time will not permit of anything like an exhaustive survey of so extensive a field, but in regard to indirect remedies it may be useful to allude briefly to the general conditions which are most often causative of neuritis, and each of them also as I think of neuralgia in the restricted meaning of undue sensitiveness of a central nature.

1. It will clear the ground a little to mention one condition, which is the most frequent and important source of neuralgia proper, but which by implied definition is never the cause of peripheral organic lesions—I mean *hysteria*. I mention this in order to dismiss it. The subject is too large, but the efficacy of hysteria in causing pain must never be forgotten.

2. *Anæmia*, too, is frequently at the root of these painful states, both central and peripheral, and the treatment is emphatically that of the general

condition. Upon this head I would mention Blaud's pill as a remedy of surpassing excellence, and especially in the form of the bi-palatinoids recently come into use. An anæmic neuritis is common. This fact is important as bearing upon the indications for the use of arsenic. I should not myself employ arsenic where I knew that a degenerative state of peripheral nerves existed, and I should endeavour to eliminate this condition by a careful examination before administering the drug for neuralgia. In anæmic neuralgia it is very efficacious. The constant association of constipation with anæmia in young women is a fact of the first importance. The late Sir Andrew Clark very ably pointed out the consequences and the danger arising from the absorption of products retained in the intestine. This is one of those (3) *toxæmic* states which are known to cause neuritis and neuralgic pains, and the underlying agency often co-operates with anæmia. Such purgatives as aloes combined with nux vomica, and the frequent administration of senna, especially in the form of compound liquorice powder, are often an indispensable adjunct to the use of iron and arsenic.

4. *Rheumatism* needs but to be recognised as a cause of nerve pain ; the use of salicylates and alkaline drugs will then suggest itself. A neuritis dependent on rheumatism seems chiefly to select the sensory nerves. What is often called " muscular rheumatism " will sometimes repay the trouble of investigation. A young man was lately brought to me because he had the impression that his liver was out of order, as indeed it was. He mentioned incidentally that he had had muscular rheumatism for some days in the calves and forearms, and tingling and numbness in the fingers. His knee-jerks were present ; there was no foot-drop, and he denied alcoholism. Nevertheless, I gave the opinion that the case was one of alcoholic neuritis. Within a week his legs were completely paralyzed, the knee-jerks were gone, and he is now recovering from a very severe and extensive attack of alcoholic pseudo-tabes. " Muscular rheumatism " is very often alcoholic neuritis, and unless taken in time, not seldom entails danger of death.

5. *Gout.* What we call the gouty diathesis is very often associated with inflammation of the nerve sheaths and degeneration of the nerves themselves. It is a particularly common cause of sciatic neuritis. This is not to say that uric acid is the toxic agent. It may be so. More likely its presence in the blood and tissues is but one among many details of malnutrition. If this be thought probable it will suggest reflections that skilful hands can turn to practical account in treatment. The pains with which we are dealing here are found most often in persons who have never had an acute attack of gout, but whose urine generally deposits lithates in abundance. There is, of course, a host of well-known therapeutic measures. Besides mercurials and salicylates I would mention colchicin in gr. 1–30 doses, lithium in the form of the bromide and the free administration of Pithcaithlie water, to which carbonate of lithium has been added. There is no need to dwell upon the importance of diet. There are, however, two other drugs which deserve special mention here. The first is chloride of ammonium. Long vaunted as almost a specific for neuralgia—and this especially in Germany—it is probable that it derives its value from its action as a hepatic stimulant. It is certainly most useful in those whose metabolism is imperfect, and who are often spoken of as gouty. Given to such persons in large doses—20 gr.—it has a wonderful power to relieve pain. The other drug is piperazin. I regard this as one of the most useful of modern acquisitions. We are all familiar with it as a solvent of uric acid, but for the relief of obscure pains in gouty people it is often indispensable. I would instance especially a form of enteralgia, probably dependent upon duodenal dyspepsia, and common in gouty subjects. It is a most painful and intractable affection, but to piperazin (gr. v.), combined with alkalis and carminatives, it seldom fails to yield.

The consideration of therapeutic measures for the relief of nerve pains in the gouty naturally suggests the subject of poisoning by (6) *lead* and some other metals. The association of plumbism with gout is a fact of great practical importance. Neuritis due to lead poisoning is not often the cause of severe peripheral pain. It is, on the other hand, productive of extremely severe and obstinate paralysis. This, however, is often less extensive than it seems, for the subjects of lead palsy are very apt to suffer from hysteria, especially from male hysteria. I have now under my care a youth of 19 years of age, a compositor by trade, who has suffered from colic. There is slight wrist-drop, which electrical examination proves to be of organic origin, but in addition there is extensive paralysis of the arms and legs, and weakness of the back. The patient cannot walk or sit upright,

and is greatly wasted. He is of an emotional temperament, has *clavus* and one-sided buccal anæsthesia, and the major part of his symptoms is undoubtedly hysterical. It would be a mistake to overlook the coincidence, and it is common. I have long had doubts as to the efficacy of iodide of potassium in lead poisoning; and Dr. Dixon Mann has lately published observations from which it would appear that this treatment is without the effect ascribed to it. He failed to find that it promoted the elimination of lead in the urine. If this be so its administration cannot be justified on other grounds, and the less it is used the better. I would suggest that in a great number of cases the best line of treatment will be to combat the gouty tendency. We are concerned here with pain only, and the characteristic pain is visceral hyp. lead colic. For that I know no remedy so likely to be useful as piperazin, combined with opium or chlorodyne, and in appropriate cases helped by a mercurial purge.

7. *Malaria* has become uncommon in England, but it is a fact worth recalling that it was at one time—and that not so long ago—widespread, almost universal. Under those circumstances we can understand how quinine—a specific for ague —came to be considered also as a specific for neuralgia. The observations of Laveran, which have disclosed the remarkable life-history of the plasmodium of malaria, have shown that quinine directly destroys the life of the parasite and so cures the disorder at the same time that it relieves the symptoms. It may fairly be questioned whether, judged without prejudice, the claims of quinine as an anodyne would prove to be better than those of any other better alkaloid. Since the recent epidemics of influenza, however, cases of frontal headache resembling brow-ague and other pains, characterised by a decided periodicity, have been very often met with, and such pains have been found to yield to quinine. There are points of analogy between the two disorders, malaria and influenza, which may make this result intelligible. In particular both seem to affect the constitution and to modify symptoms permanently or for a long time. Thus, it is likely that, for the present, quinine will, to some extent, vindicate its reputation as an agent for the relief of pain.

8. I have already cited a case of neuritis due to *syphilis*. Of the treatment in such a case I need not speak.

9. *Diabetes* as a cause of neuritis is well recog-

nized. I have urged the probability that it gives rise to neuralgia also. In one of my beds at present is a boy who, when first seen, had a curious rhythmical pronator spasm of one hand. There was history of a fall, to which no importance was attached. Soon were superadded momentary fits of aphasia, resembling *petit mal,* and finally severe facial neuralgia. On admission his urine was found to be loaded with sugar.

My purpose in recapitulating these better known causes of nerve pain has been to suggest rather than to indicate a vast number of remedies which it is impossible to mention. In conclusion, I would dwell upon the necessity of bearing in mind that more than one cause and more than one toxic agent may be present in any given case. Instances of this have been cited. Most important of all it is to remember that, with the nutritive functions impaired, and the eliminatory channels blocked, the accumulation of poisons in small quantities may be disastrously effective. I was asked to see a lady thought to be moribund. There was an alcoholic history. The patient had a cirrhotic liver and ascites; and the blockage of the portal circulation was still further evidenced by hæmorrhoids. There was stertorous breathing, unconsciousness, coma by day, and violent delirium at night. Respiration and the heart's action were good. The patient was receiving three ounces of brandy to maintain her strength. Alcoholic paralysis was declared by foot and wrist drop, and extreme tenderness of the extensor muscles. Stimulants were withheld, and she made a temporary recovery. It is probable that with the tissues already saturated with alcohol, and the function of the abdominal viscera suspended, even so small a quantity of the poison sufficed to produce alarming symptoms. I need not enforce the doctrine that the state of the system, and especially of the emunctories, should in all cases engage the attention of the physician.

Of the direct anodynes time will not permit me to speak, nor could my experience of their use add anything to the sum of knowledge already so great. It may be said, however, that the activity of synthetical chemistry in constantly supplying new drugs of this class may have the disadvantage of tending to induce forgetfulness of the older tried remedies, of which opium holds the highest place.

Local treatment of neuritis. It has been said that a large proportion of the painful states now

under consideration depend upon the pathological state called neuritis. This cause is especially frequent where an extremity is the seat of pain. Neuritis is a local condition, and local treatment is of the first importance. A case of acute sciatic neuritis may be taken to illustrate this. A cause has first to be sought and can usually be found. It may be a blood state, as gout or diabetes, or it may be a lesion such as sacro-iliac disease or some other, which causes pressure upon the sciatic nerve within or outside the pelvis. Whatever the cause, concurrently with the measures taken to obviate this, something must be done to relieve pain and to promote the restoration of the nerve to its normal state.

By far the most important of local remedies is *rest*. Upon this I need not dilate, but will only say that to find and maintain the position of greatest rest—a difficult thing in itself—will repay the utmost care. A uniform *warmth* of the part should be maintained, and this is best done by swathing the limb in sheets of cotton—or pinewood —wool, lightly maintained by a loose flannel bandage, and the whole arranged in such a way that the necessary local applications can be effected with as little exposure as possible. Occasional hot *fomentations* relieve pain. *Counter-irritation* is of the greatest value. This may be achieved in many ways. *Sinapisms* in the course of the nerve are very useful. They may be small, and two or three or even more should be applied at a time at a distance from each other, and the place of application changed, so as to produce and maintain a constant redness of the entire surface of the skin overlying the sciatic nerve. Great relief is obtained in this way—much more, I think, than by cauterisation. A *blister* may be called for. The inflammation in the sheaths of the nerve is constantly varying, and its seat moves about. If at any point where the nerve is comparatively superficial tenderness and swelling can be felt—a blister applied for some hours near this point is likely to have a rapid effect. Such swelling may sometimes be felt and even seen beneath the head of the fibula, where the external popliteal nerve approaches the surface. The question of administering opium or some other *anodyne* drug will arise. Upon that point the physician will use his discretion, and will probably have his favourite remedy. But when all necessary weight is given to the objections to its use in a given case, the potential excellence of opium must never be forgotten.

Upon this head I would merely mention that pain is largely a habit, and to break that habit is the best way to cure the pain. Any one of many drugs will help towards that; but where there is severe and extensive inflammation in the sheaths the local sedative action of opium is invaluable. By this local action is meant the effect locally when the drug is taken internally. The local application of laudanum, or of powdered opium upon poultices and fomentations, so often adopted, seems indeed to be warranted by experience, but its utility is hard to account for.

The injection of cocaine into the buttock is often attended with prolonged relief. It is useful in breaking the painful habit, and it further tends to reduce inflammation by diminishing the blood supply. Cocaine is thus of great service, but it must be used with caution. Idiosyncrasy is here an important factor. I have twice produced alarming symptoms by the injection of less than half a grain, and yet a very high authority, writing in a well-known work of reference, counsels the use of so much as a grain. My experience would deter me from this.

I come now to speak of the *electrical* treatment of neuritis. There is in the galvanic current an expedient for the control of pain, of which unhappily too little is known. Those who have had occasion to use it are well aware of this in the case of neuralgic pains of obscure nature: but I would advocate its use daily and persistently in the earliest stages of acute neuritis. The current judiciously used has the power to ease pain and stay the degeneration of implicated muscles. I have satisfied myself of this over and over again, and I believe that electrical treatment finds just here in these acute peripheral conditions its most valuable application. Experience enables me to disregard the confused idea which prevails that an electrical current is necessarily an irritant to inflamed nerves, but indeed I have never shared that view even in theory, for the reason that there is no such thing as an inflamed nerve at all. To excite the function of a nerve cannot conceivably excite inflammation in its sheath, which may indeed be inflamed. And if the nerve proper be degenerated its function cannot by any means be excited. That of the nerve endings, however, can, and whether in this way, or in some other, muscles that would inevitably have wasted can be made to retain their nutrition, degeneration in the nerve can be arrested, and pain can often be controlled ab-

solutely. The application should generally be made with the positive pole, and the tender points of the limb as well as the degenerating muscles, and anæsthetic areas, should be attended to.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

January 29th, 1895.

A Case of Severe Tophaceous Gout.

A man, aged 58, brought by Dr. Sturrock from the St. Pancras Sick Asylum, offered an excellent example of gout with large chalk-stones. The tophi were situated on his knuckles, fingers, and over his elbows. Both knees were somewhat stiff and creaked on movement, and his right ankle was ankylosed. He was grey and looked older than his years, and was of a pale complexion. He had been a fruit-dealer, and had lived much in the Channel Islands. He said that his father and his father's brother had both suffered from gout, and that they had both had two sons, of whom the younger only, in each instance, was the one to inherit the malady. He had himself had many attacks of gout and from no fewer than nine of rheumatic fever. These latter, he said, had been considered to be quite ordinary attacks, and had usually lasted about six weeks, and resulted in complete recovery. He had had an abscess by the side of the ankle-joint, which had resulted in its ankylosis. On examining his knees distinct and large lips could easily be traced along the margins of the condyles.

Comments. It would not be easy to produce a better case in illustration of the assertion that gout is usually an appanage of rheumatism. The term rheumatic gout is well justified as implying a mixture of the two affections, but the facts would be more correctly expressed if we were to speak of gouty rheumatism. It is the tendency to rheumatism which is fundamental and the gout which is superadded. Thousands of patients have rheumatism without gout; but it is rare indeed to find a patient suffering from gout who is not rheumatic also. You have heard this patient's statement as to his nine attacks of rheumatic fever, and you have recognized in his knees and some other joints precisely the changes characteristic of the so-called rheumatoid arthritis, yet these are coincident conditions with a large development of chalk-stones. Now, it is very common for the children of gouty parents to suffer from rheumatic fever. The knowledge of this fact is not new ; it is as old as the time of Heberden. My own experience has been that whenever a patient gives a history of many attacks of rheumatic fever there is almost always gout in the family.

I am asked to speak as to the treatment of gout. That is a very long subject. I am, of course, a believer in dietetic rules. They are paramount. During the attacks aconite, colchicum, alkalies and quinine are helpful. Both during the attacks and between them it is of primary importance to supply the blood with plenty of fluid so as to favour elimination by the kidneys. I am a great believer in the value of tea as preventive of gout, and as aiding in the elimination of uric acid. It should be taken weak and quite freshly made. I also always insist on the avoidance of sweet fruits and on the free use of green vegetables.

An exceptional form of Vest-eruption allied to Lichen Circumscriptus.

A young man of 24, sent by Dr. W. Walker, presented a good example of a lichen eruption on the chest and trunk, due, in all probability, to the irritation of his vest. He said he had suffered from it for three or four years, but that it had been worse of late. The eruption was very plentiful and did not take exactly the form usually presented by lichen circumscriptus. Many of the patches were very large and more or less ringed and gyrate, but there were a great number of quite discrete, florid, acuminate lichen spots. There was no eruption on other parts of the body, excepting a little tendency to florid acne on the face.

Comments. The eruptions which we may conveniently know as " Vest-eruptions " assume a great variety of forms, ranging between urticarious erythema and the common lichen circumscriptus so frequently seen in the middle of the chest and back. They may occur at any time in the year, but are especially common either in very cold or very hot weather. The urticarious type usually occurs in hot weather, and is due to excessive

perspiration, whilst the lichenoid varieties are met with more frequently in winter when men put on thicker vests and wear them at night as well as in the day. The urticarious forms are usually transitory, but the lichenoid may, as in the present instance, last for years. The present is a somewhat exceptional case, on account of the great extent of the eruption and the amount of irritation present. It reminds me most of a case in which we took much interest some years ago and of which I possess two portraits, taken at distant periods. In the common lichen circumscriptus the patches are usually few in number, confined to the middle of the chest and back, and very stationary. In the case to which I refer, however, and in the present one, they cover the whole of the upper two-thirds of the trunk and undergo considerable change, both in position and general appearance at different times. The case to which I refer puzzled us very much for a long time, and no treatment seemed to do any good. I have no doubt, however, that our final conclusion that it was a vest eruption was correct. I brought the man here some months ago in order that we might compare him with his portraits, and see if he were completely cured. I have, however, a few days ago, seen him again with an acute relapse, and next week I shall, I hope, have an opportunity of showing his present condition. The return of his malady has most definitely been due to his having purchased some new woollen vests for protection during the recent cold weather. He tells us that the irritation caused by his new garments was such that he was obliged to lay aside his drawers and go back to his old ones. He persisted, however, in wearing his vest, and the sides of his chest are now covered with lichen.

A Peculiar Form of Bazin's Malady.

This patient, a girl of 14, was brought by Dr. Barratt, of St. John's Hospital. The inner sides of her knees and upper parts of legs showed a number of little round ulcers, looking, some of them, exactly as if punched out. Lower down on the legs there were numerous scars—some of them indistinct, others large and conspicuous. On one knee there was a little patch of white tallowy skin, evidently gangrenous. No subcutaneous indurations could be detected, nor was there any evidence of burrowing from one ulcer to another. The patient was pale and very feeble-looking.

Her hands and feet were dusky and cold. It was stated that her father, one grandfather, and one grandmother had all died of phthisis. One of her sisters had suffered very badly from chilblains, but the patient herself said that she had never had one in her life. In a former winter her hands and forearms had suffered, and numerous round scars, much like those on her legs, were visible about her wrists. All the scars were perfectly sound, and there was not a trace of anything like lupus anywhere.

Comments. There can be no doubt that the ulcers in this case are the combined result of a very feeble circulation and inherited tubercular tendencies. To these must be added, as an exciting cause, the local influence of cold, for the patient has told us that she gets well in summer and is worse in winter. The case illustrates well what we often have to remark—that patients with a very feeble circulation are not those most prone to chilblains. The circulation may, indeed, be too feeble to admit of the development of the latter, and such probably is the case in this instance. We have in the Museum several portraits illustrating the occurrence of multiple ulcers on the forearms and hands in alliance with strumous tendencies and feeble circulation. There is none, however, which shows, as in this patient, their combination with ulcers of the legs. It is the presence of the latter which leads me to use the designation Bazin's malady, fully admitting at the same time that our patient presents exceptional features. Let me insist, as we have done on many other occasions, that these multiple, punched-out ulcers, although at first sight very suspicious, have nothing whatever to do with syphilis.

Lupus on the Cheek, with History of a Congenital Mole or Nævus.

A girl of 14, sent by Mr. G. W. Sequeira, presented an interesting patch of lupus just in front of one ear. The patch was of irregular shape, about as large as a shilling, and very superficial. It was well characterized, and in the middle there was a small scar. It had nothing of a nævoid character, nor was there any trace of the structure of a mole; but a point of great interest in the history was that the aunt asserted that there was a red spot there at the time of the child's birth. She was an intelligent woman, and was very confident in her statement. The girl was very delicate-looking, and had a bad cough.

Comments. My theory of lupus and other tubercular affections of the skin is that they are not, in the majority of cases, caused by inoculation, but that they result from the development of parasitic elements which were previously dormant in the tissues. Thus, any damage to the vitality of the part may lead to a patch of lupus or a tubercular ulcer. Now and then the localizing lesion may be congenital, and I have long ago asked to be allowed to name a certain form of lupus "Nævus-lupus." There is nothing improbable in the suggestion that the parasite of tubercle may flourish in the structure of a nævus, and may, indeed, find it more vulnerable than the healthy skin. With this theory the present case fits well, but it differs from most which I have previously described, in that the disease is now lupus vulgaris, and shows none of the characters of an infective angeioma or "lupus-nævus."

THERAPEUTICAL NOTES.

The Treatment of Whooping-Cough with Quinine.

—Baron reports on fifty cases of whooping-cough treated after the old manner of Benz-Ungar, which they advocated in 1868. In a few children the good action of quinine was noticed in two or three days, but in most of the children the results were shown after several days. The first evidence of value is the lessening of the night attacks. The improvement continues until health is restored, unless the dose be too suddenly reduced. The author continues the administration of quinine for three weeks. Relapses do not occur in children treated with quinine. In spite of the fact that the quinine was not given as regularly as directed in more than half the cases, there were only two failures noted. The treatment is of unusual value in the cases of acute inflammation of the lungs caused by the whooping-cough. Since the thorough trial of quinine, whooping-cough has lost all its terrors to the author.

The proper dose of quinine is one-sixth of a grain for each month of a child's age, and one and a half grains for each year, given three times in the day—at 6 a.m. and 2 and 10 p.m. More than six grains three times daily is not necessary for older children. As the case improves, the number of daily doses is decreased.—(*Ber. Klin. Woch.*)

A New Method of making Milk Palatable and Digestible.

—Dr. Robert T. Edes, of Boston, gives a valuable way of preparing milk where other methods have not proved useful:—A pint of milk is gently warmed. Into it is dropped, very slowly and with constant stirring, about 20 minims of the dilute hydrochloric acid of the United States Pharmacopœia. The milk should be stirred until it cools. In this way a very fine flocculent coagulum is produced, floating in the whey, which is easily accessible to the digestive secretions, while the whole fluid has lost somewhat of the flat and cloying taste which makes it unacceptable to so many. It will be noticed that milk prepared in this way differs from the various "wheys" in the highly important particular that the casein is retained and used, instead of being separated out as a distinct product, while it avoids the bitterness of pancreatinized milk.—(*Bos. Med. and Surg. Jour.*)

Hiccough Treated with Tobacco Snuff.

—Dr. G. Tatevosoff draws attention to the excellent service which may be obtained from the ordinary snuff-tobacco as a means for cutting short hiccough. He relates an instructive case of a patient with some chronic chest disease, accompanied by violent cough attacks, in whom the latter used to be followed by extremely obstinate hiccough. The common remedies (including cocaine) failing to exercise any controlling influence on this most distressing symptom, Dr. Tatevosoff at last decided to give a trial to the said old-fashioned popular means, making the patient on each occasion thoroughly snuff into his nose a pinchful of the powder until the appearance of a lively sneezing. From the first *seance* "the effect was truly brilliant, the hiccough subsiding as if by magic." Under the influence of the simple remedy the attacks steadily became milder, and ultimately vanished, though the patient's cough remained as intractable as ever.—(*St. Louis Med. Surg. Jour.*)

To Hide the Taste of Chloral.

—Dr. E. Holland calls attention to the fact that the taste of chloral hydrate is effectively masked by lemonade. Two or three drachms of the syrup should be placed in a tumbler with about two ounces of water. If to this is added about two ounces or so of gaseous (bottled) lemonade, the mixture may be drunk at leisure, and the soporific action of the drug is in no way impaired.—(*Med. Bull.*)

A CLINICAL LECTURE

ON

FRACTURE OF THE LONG BONES FROM SLIGHT CAUSES

(Sometimes improperly called "Spontaneous" Fracture).

Delivered at S⸱. George's Hospital, January 22, 1895, by

WILLIAM H. BENNETT, F.R.C.S. Eng.,

Surgeon to the Hospital, and Examiner in Surgery
at Cambridge University.

GENTLEMEN,—I suppose most of you have learnt by this time that, speaking broadly, the immediate importance of a fracture is dependent, to a great extent, upon the amount of force which is concerned in its production. This naturally must be so, seeing that the greater the violence the more abrupt would be the break, and the greater must be the damage sustained by the soft parts about the bone, and the more severe the shock to the patient. Nevertheless, it happens that there are met with, frequently enough to be of considerable practical interest, fractures of long bones produced by very slight causes, some of which are the most important, clinically, with which we, as surgeons, have to deal. Patients whose bones break easily in this way are nearly always found to be the subjects of some general or local disease or idiosyncrasy, with which the tendency to breakage from merely slight injury is connected. Such being the case, the occurrence of a fracture which seems to have been produced by an amount of violence which ought not, under ordinary circumstances, to be sufficient to cause the breakage, should arouse suspicion and lead to a careful investigation of the case with a view to ascertaining whether one of the various conditions which tend to produce undue brittleness of the bones is present.

There is a woman now in No. 7 bed, Wellington Ward, about 56 years of age, who is a very stout subject. So far as she knows she has been well for many years, excepting that from time to time she has had some little rheumatic pain in her lower limbs. The night before she came to the hospital she felt weak, or at all events tired, and in order to get into bed raised one limb by placing the hands under the thigh, just as many people will do who are tired, weak, or very stout. As she lifted the thigh in this fashion she felt the bone give way—she had broken her thigh. That is an instance of fracture from a slight cause. In normal conditions under no circumstances could she have produced a fracture of the femur by lifting the limb in that way; the femur was in her case too weak to bear the strain of lifting the leg. Therefore, when I saw the patient after her arrival at the hospital, I strongly suspected that there was at the bottom of the case some abnormal condition not necessarily confined to the bone itself which accounted for the peculiar ease with which the bone was broken. So I began to cast about for a cause.

Now, the principal conditions which tend to easy fracture are the following :—

1. GENERAL, *i.e.*, when the tendency to fracture is secondary to some predisposing disease or peculiarity, not limited to the affected bone :—

> Tabes (Locomotor Ataxy).
> Rickets.
> Mollities Ossium.
> " Fragilitas " Ossium.
> General Fatty Degeneration.
> Chronic Lead Poisoning.
> Malignant Disease.

2. LOCAL, *i.e.*, when the influences leading to the tendency to easy breakage are limited to the bone itself and are independent of other co-existent disease—

> Bone Atrophy from long disuse.
>   "   "   from injury to the Nutrient Artery (?).
>   "   "   in old age (intracapsular fracture of femur).
> Weakness of the bone produced by necrosis or caries.
> Malignant Disease.

3. MUSCULAR ACTION alone may cause fracture under certain circumstances; to fractures thus arising the term *spontaneous*, if it is used at all, should be limited.

Having the suspicion just referred to with

regard to the case, I began to examine the patient for one of these causes. I could find no history of locomotor ataxy, nor of any other of these causes, except, perhaps, fatty degeneration, as the woman was very stout. In certain cases where the subjects are very fat the compact tissue of the bones may be gradually replaced by fat, which is continuous with the medulla of the bone. So much does this happen sometimes that the compact tissue becomes very nearly as thin as a sheet of writing-paper. The result is that any very slight force may produce a fracture of such a bone. Still there was no reason to think that this woman was suffering from such extreme fatty change as that. Inquiries as to whether she had any lump or swelling about her led to the reply that, so far as she knew, she had not. Knowing how curious people sometimes are in this respect, and being aware how fond they are of concealing or attaching no importance to certain diseases from which they suffer, I said "Have you no swelling in the breast, or anything of that sort?" "Oh, yes," she said, "I have a lump in my breast, but I have had it for years; that's nothing." However, upon exposing this lump, which was in the right breast, it proved to be a hard tumour the size of a small orange. We had thus obtained the key to the situation; this woman was the subject of scirrhous mammæ, and the femur had become secondarily involved by malignant disease. The medullary tissue had become infiltrated by the disease, the compact tissue had been thinned down so much that the lifting of the thigh with the hands was sufficient to break the bone. This, therefore, was a case in which fracture from a slight cause was the result of malignant disease of the bone, secondary to that in the breast. That was my interpretation of the case.

The important question then arose as to what should be done for this poor woman. Here was a patient with a fracture of the thigh and a malignant tumour in her breast. The tumour could easily have been removed, but, supposing that this condition of the thigh was due to cancerous disease, it would have been mere cruelty to remove an obvious cancer in the breast which caused no trouble, leaving behind a bone, the seat of the same disease, which was secondary in character. Such a course could not be entertained, for it would be contrary to sound surgical practice under the circumstances. Later on, however, when the thigh, having been treated in the ordinary way,

had settled down and become more or less comfortable, I had a consultation as to what should be done for it, and it was very properly decided that surgical interference was unjustifiable. In my own mind I had not the least doubt as to the condition of the thigh, but it so happened at the date fixed for the consultation that the fracture had become more or less set—some union seemed to have taken place. It was, therefore, suggested that this might not, after all, be a case of malignant disease of the thigh bone, as union was taking place, because union after a fracture from malignant disease is not an ordinary occurrence. That is quite true, but temporary union after fracture connected with malignant disease does sometimes occur; and this is a point to which I wish particularly to call attention. There is a specimen in our museum of the union of a fractured femur in which there was malignant disease, as has apparently happened in this case. The history of the museum specimen illustrates very well the clinical course of some of these cases of fracture. The case occurred as long ago as 1859, and was reported in the Pathological Society's Transactions by Mr. Holmes. This is part of the description which is given, from which you will see how unexpectedly interesting some of these cases turn out to be. The patient was a woman, 52 years of age, who was admitted into the hospital as a physician's patient, complaining of pain—which she had been subject to for three years—in the region of the left hip. It was at first supposed to be rheumatism—the majority of these cases are at first supposed to be rheumatic—and mercury was given her (as was the fashion in those days) until her gums were affected. As, however, she continued to get worse, the surgeon was requested to see her. Whilst he was manipulating the thigh, for the purpose of examination, it gave way, and the lower fragment started up almost under the skin.

Now, this history is fairly typical of many of these cases of fracture from slight causes, and the case itself is not unlike the one of the woman about whom I have been speaking. There was a growth of cancer in the femur, which had so absorbed the central portions of the bone that there was not enough compact tissue left to allow the limb to be lifted off the bed without breaking from the leverage exerted by the weight of the distal portion. The main reason, however, for mentioning this case—reported as far back as 1859—

is that the specimen shows that union after malignant disease does sometimes happen, and union in connection with fracture after malignant disease has, perhaps, occurred in the patient in the Wellington Ward. Existence of cancer is, however, not negatived by this union if it has occurred, for already there is a large mass growing around the seat of the fracture, and before long any union which may have taken place will probably give way again in consequence of absorption of the uniting medium by the malignant disease. This case, then, will show you the importance of always making a careful general examination of a patient who is the subject of a fracture from a very slight cause; and it apparently shows the possibility of temporary union after fracture from malignant disease—two very important points.

There was in the surgery, two or three days ago, a man who was under my care in the hospital in August, 1894. In going downstairs with a heavy weight on his shoulders, he slipped off the last step. He did not fall, but stumbled, yet he found, on trying to put his weight on his left leg, that the limb had broken. Now, in a general way, such a cause will not account for the breakage of both bones in the leg of a healthy man. It is the kind of accident which will sometimes fracture the fibula, but not the tibia with it. I therefore went into the general state of this patient, and found he was the subject of locomotor ataxy, which was the key to the situation in this case. Fractures in patients who suffer from tabes are very interesting, and at the same time very important from a clinical point of view. As I have mentioned, in fracture of a bone which is infiltrated with malignant disease, union is of the rarest occurrence and only temporary. In patients who are the subjects of tabes, on the contrary, the bones unite very quickly, but not altogether soundly; they also generally unite in a somewhat peculiar fashion. You will find in nearly all these cases that the amount of callus thrown out round the bone is excessive; so much so sometimes that, at the end of ten days or a fortnight, especially if any movement in the fracture has taken place, there is a mass as large as a closed fist around the break. It may even look like a large isolated rounded tumour about the bone. This excessive bone formation is not merely callus such as is thrown out in ordinary fractures, but is to a great extent due to ossification in the soft parts about the injury. This may occur to even such a degree that

the muscles and the other soft tissues may sometimes become so solid that the whole thickness of the limb at the level of the break has the appearance of being one solid mass of bone. This condition is exactly akin to the arthropathic changes which are found in "Charcot's joints," in certain persons who suffer from locomotor ataxy. It is not merely an alteration in the shape of the bones, it is an addition to the bones, really, of bony tissue at the expense of the soft parts round the fracture or articulation. All fractures in tabetic people require more care to be taken in keeping the bones quite still and absolutely fixed for a long period than is necessary in people who are healthy, in the ordinary sense of the term, on account of the tendency of the union to soften; for, as I have already said, the union in these cases, although rapid, is not firm. At first the repair seems perfect; but directly the patient discards splints and begins to bear weight on the limb there is a considerable probability that the bone will bend at the seat of fracture. That is my reason for mentioning the case just described. The fracture united perfectly, and when the patient left the hospital everything seemed strong and sound. After a considerable period, as much as four months, he was allowed to leave off the splint, but when he began to walk on the limb it bent at the point of injury.

This reminds me, in passing, of one other little point of interest in these tabetic fractures. It is an old-fashioned superstition—and, on the whole, I think it is founded on fact—that in a case of ordinary fracture, if the bone is broken again the second fracture very rarely happens through the seat of the former break. That is because, in a general way, if the bones are in proper apposition (the remark, therefore, only applies to fractures which have been properly "set") the medullary canal is very frequently obliterated altogether and replaced by compact bone, so that the bone is stronger at the seat of the old fracture (because it is solid) than it is above or below it. Now, in fracture from slight causes, whether the patient be tabetic or not, if the limb is broken again, the fracture is generally through the old union—a fact which shows how deceptive the apparent soundness of the union may be in such cases.

The manner in which the hypertrophic changes I have been describing may be produced by free movement between the ends of the fractured bones in tabes is well illustrated by the case of

J. J., a patient of mine, aged 40, now in the Oxford Ward. The man, who was a fairly intelligent Welshman, had, nine months before coming to the hospital, sustained an injury to the lower part of the right leg. Following the custom prevalent in his part of the country he went to a bone-setter, who found some "small bones out of place," and with a view to replacing them he manipulated the injured part violently. This proceeding was repeated several times at intervals without doing any good; indeed, the parts, on the contrary, became very painful and gradually swelled to a great size. Some time previously the patient had sustained a slight injury to one forearm which was treated by the same bone-setter in much the same way, with the result that a bossy swelling formed over the injured bones, and permanent weakness of the forearm followed. On his admission the lower third of the right leg was swollen in the form of a hardish bossy mass of the size of a large cocoa-nut; below this the ankle was distorted, apparently disorganized, and on both sides of it were fluctuating areas. There was no tenderness on pressure, but in the bossy part of the swelling there was continual distressing pain. In the middle of the left forearm was a badly united fracture surmounted by bossy thickenings. The man was the subject of tabes. The pain was so acute that amputation of the affected leg was decided upon, and subsequently performed just below the knee. The stump healed well, as is the usual habit in such cases. Upon examination after removal it was found that there had been a fracture of both tibia and fibula above the ankle. There was no union between the bones, but all around the fracture were large irregular masses of bone, partly amongst the tendons and partly under the skin, involving, in fact, all the soft parts, more or less. The ankle-joint was disorganized and suppurating. It is quite certain that these changes were caused by the energetic movements employed by the bone-setter, which, in a healthy subject, although they may have been disastrous, would not have led to the production of these large osteophytic masses of bone which were the outcome of this patient's tabetic state.

The following is a phenomenal illustration of multiple fractures from slight causes, the brittleness of the bones depending upon a malady which is hardly recognized as it should be as a cause of this condition. The patient was, when I first saw her, about 45 years of age. She had then recently taken a house in London, and the first time she entered it she slipped on the parquetry flooring of the hall, and sat rather than fell down, and thus broke both bones of her left leg about three inches below the knee. The fracture was absolutely transverse—an important point in these fractures from slight causes to which I shall again refer. When I saw her I took into consideration the ease with which the fracture appeared to have been produced, its situation, and the fact that it was quite transverse, and felt certain on these accounts that there was something peculiar about the case. My catechism of the patient, however, elicited nothing material, and as I was not then so experienced as I am now, I thought I had perhaps made a mistake, and that after all the case was of an ordinary kind. I put the fracture up in plaster of Paris; it healed admirably, and I heard nothing more of the patient until about six months afterwards, when in getting out of her bath, as she put her right foot on the floor "something gave way" in the limb, and she fell to the ground; she had broken her right leg in the same situation as the first fracture in the opposite limb. This second break corroborated my previous idea, especially as this fracture was also absolutely transverse, and had been caused by a very slight injury. Again I tried to find out what was at the bottom of the case, but all that I could ascertain was that her muscles were a little weak. Therefore, again rather at a loss, I put up the fracture, and it healed very well. I heard nothing more of her for nearly a year, when I was sent for to her, as she had broken her thigh in attempting to rise from an easy chair. She subsequently broke one leg again about nine months afterwards. Although I attended this patient within four or five years for these multiple injuries of the lower extremities, it was not until after the third fracture, when I noticed she was suffering from slight double "drop-wrist," that I ascertained the cause of her brittleness. It appeared she had been in the habit for many years of taking medicine containing a considerable amount of lead, and had also regularly used an application for the hair which was very fashionable in those days, and contained large quantities of lead. In point of fact this patient was clearly the subject of extremely chronic lead poisoning, and at the time of the last fracture she had so little power left in her muscles that she was either carried or led about by two people, one standing on each side of her.

The key to the tendency to extreme fragility of the bones in this case, so far as I could ascertain, and so far as the nerve specialists who saw her could tell, was chronic lead poisoning. Doubtless some of you have learned that people who dabble in lead, such as house-painters, are more prone to suffer from delayed union after fracture than any other class of the community.

I have often pointed out this fact in the wards, and when I have under my care a patient suffering from fracture, who is concerned, in his work or in other ways, with lead, I always allow him a fortnight, three weeks, or, perhaps, even a month longer to bring about a thoroughly sound union than I should give another man whose occupation did not bring him into contact with lead. This is a good practical point, which is learned by experience, and is extremely useful.

As I have mentioned, the fractures of this patient's bones healed firmly and well, with no unusual amount of callus. In these cases of fractures from slight causes the direction of the break is always transverse. This is an important symptom from a diagnostic point of view, and a fortunate one in the matter of treatment, as transverse fractures are, of course, the easiest of all to manage, for, when once the bones have been placed in position, there is seldom the least difficulty in treating the case, as there is not the same tendency to displacement as there exists in oblique fractures; the bones, in fact, keep in position almost of their own accord. This, doubtless, accounts to some extent for the rapidity of the union in many of these cases.

I have mentioned that people who are extremely stout—those, in fact, who may be said to be subjects of general fatty degeneration—are prone to meet with fractures from slight causes. You can easily imagine that that would be so, but it is not always easy to produce actual proof of the fact. The following case is, therefore, worth relating:—

It was about three years ago that an enormously stout patient came into Princess Ward under my care. She had been suffering for a very long time from chronic œdema and ulceration about the left leg, above the ankle. The disease was clearly incurable by ordinary methods, and, after consultation with my colleagues, I amputated this poor woman's limb just above the knee. We found at the operation that the thigh was simply one mass of fat; of muscular tissue scarcely anything could be seen at all. The compact tissue of

the femur was a mere shell—it could not have been thicker than a piece of thin card-board—so that the saw cut through it almost as if it had been cheese. She did quite well, and the stump healed perfectly. She was about to leave the hospital, but before doing so was, as usual, being taught to use crutches. In attempting to use the crutches on one occasion she seemed about to slip, and a nurse or one of the other patients prevented her from falling, but in so doing made some pressure on the stump. The woman, thereupon, gave a cry of pain, and fell back on the bed. It was found that her thigh had given way just below the great trochanter. This shows how slight a cause may produce fracture in such cases of fatty degeneration. No union followed in this instance, and the part became flail-like. Such is the result which follows in fracture from fatty degeneration, as there is really not enough bony tissue to produce the uniting medium.

It is not very unusual to hear of fractures from muscular action, but they are seldom seen in practice, except of course in the case of the patella, fracture of which almost always arises from muscular action, but it is not the kind of injury I am now dealing with; the remarks, therefore, which I am now about to make apply only to the long bones. Fractures from muscular violence *alone* are, I believe, very rare, but they undoubtedly sometimes happen, and the following question arises—Does a fracture of a long bone from purely muscular action ever happen in a person whose bones are as strong as those of ordinary people? For my own part I very much doubt whether such fractures occur in people whose bones are quite natural, although they do happen in people in whom no disease or defect is obvious, and who, for all intents and purposes, are in a general way apparently strong and well. Here is a case in point, which occurred in my own practice.

A woman of about 47 years of age was walking along the pavement, when she trod on some slippery substance. To save herself from falling she stretched out her hand to catch at the area railings, but before she could reach them someone caught her and prevented her from coming to the ground. She did not touch the railings, but the violence of the action of throwing out her arm was sufficient to break her humerus; the fracture, as is always the case, was completely transverse and situated just below the insertion of the deltoid. That, then, is a case of fracture from muscular

action pure and simple, and it is to that sort of case that I consider the term "spontaneous fracture" should be limited, if it is used at all.

The main reason I have in mentioning this case is that it seems to be an instance which corroborates my idea that people who suffer from such fractures are not quite normal in their bones. This fracture united perfectly well, and was very easily managed, because it was transverse. I heard nothing more of this patient for, I should think, nearly two years. She had then, I was told, broken her thigh in the middle by catching her foot in some obstacle as she was walking.

This, I think, showed pretty clearly that some peculiarity existed about this patient which led to her bones being very brittle, but simple brittleness of the bones does not amount to anything like a disease. There are certain people who are perfectly healthy and perfectly strong in every other respect, but whose bones, without doubt, break more easily than those of some of their fellows. What the reason of this is I do not know; the bones seem normal in size, and the patients seem to be strong and well developed. It is to cases like that, I take it, that fracture from muscular action alone is generally almost entirely limited. To such cases in which no local or general defect is apparent the term "fragilitas ossium" may be properly applied.

Time does not now permit of my giving examples of fractures from slight causes arising from the other conditions which I have tabulated, although they are all of much interest.

I would impress upon you, in conclusion, the following practical deductions :—

I. Fractures from slight causes connected with :
   (a) Tabes and locomotor ataxy—unite rapidly but not strongly.
   (b) Rickets, fragilitas ossium, and lead poisoning—unite well but sometimes slowly.
   (c) Malignant disease—very rarely unite at all, and the union if it occurs soon breaks down again.
   (d) Mollities ossium and general fatty degeneration—do not unite at all.
   (e) Fractures of long bones caused by muscular action alone — unite well and strongly.

II. Fractures from slight causes are for practical purposes always transverse; they are, therefore, very easily managed.

III. All cases of fractures, in which the violence described appears inadequate for the production of the lesion, should be regarded with suspicion, and a very careful general examination of the patient made, in order to ascertain whether either of the conditions enumerated above is present.

IV. Fractures from slight causes are rarely if ever seen in limbs affected by infantile paralysis, or other forms of palsy connected with disease of the brain or spinal cord. Seeing the influence which disease of the nervous system undoubtedly has upon the tendency to fracture, this is, at first sight, somewhat remarkable; but that such is the fact I have no doubt, although when, on one occasion at the Medical and Chirurgical Society, I made the statement, it was traversed by a distinguished surgeon who was present. Fractures from ordinary violence occur, of course, in paralyzed limbs, but in my own experience, which is considerable, I have never seen a fracture from a "slight cause" in limbs so affected, and the experience of several surgeons whom I have questioned upon the subject is the same as mine. Moreover, a search through the records of St. George's Hospital extending over a quarter of a century has not afforded a single instance of the fracture of a paralyzed limb of the kind mentioned from a "slight cause"; although in limbs which have become partly paralyzed in consequence of injury to the nerves passing into them, such fractures do occasionally happen. For this there is, I think, a possible explanation, but I do not propose to go into that question at present.

A POST-GRADUATE LECTURE
ON THE
TREATMENT OF SOME COMMON VULVAR AFFECTIONS.

Delivered at Charing Cross Hospital, Feb. 7, 1895, by
AMAND ROUTH, M.D., M.R.C.P.
Obstetric Physician to the Hospital, with care of Out-Patients.

IN a post-graduate lecture delivered at this hospital on October 25th last, and subsequently published,* the various affections of the vulva were discussed as they occur in the three stages of life—before puberty, during menstrual life, and after the menopause.

* *Clinical Journal,* December 5th, 1894.

The subject was dealt with more from the etiological point of view, and it was pointed out that each of these three periods had certain vulvar affections almost peculiar to them, owing to the risks and predispositions to which those periods of life are liable.

I propose to pick out from the large mass of vulvar diseases a few of the more important ones, and to speak mainly of the treatment which I have found most useful.

Vulvitis. Before puberty and after the menopause the genital organs are not endowed with much functional activity, and thus a vulvitis is less apt to spread along to other structures, which, though anatomically continuous, are not physiologically identical. After puberty—between that date and the cessation of menstrual life—the vulva is, anatomically and physiologically, continuous with important organs in several directions—viz., with the numerous glands and follicles on its surface-membrane, especially with the glands of Bartholini; with the urethra also, and the many glands opening into the floor of its canal, especially the two ducts or glands of Skene, and, most important of all, with the genital track, from the vaginal orifice to the fimbriated ends of the Fallopian tubes, and on into the peritoneal cavity itself.

These facts explain how it is that the vulvitis of infancy usually remains a vulvitis, rarely extending to the vagina, the hymen being a sufficient anatomical barrier.

For the same reasons, senile vulvitis, even though often of constitutional origin, rarely spreads along the genital canal.

Between puberty and the menopause, however, the sequence of events in vulvitis is quite different, and, as we saw in the last lecture, it will spread to the above-named glands, causing sometimes a large retention cyst of the gland of Bartholini, which may suppurate; or it may spread to the urethra, and lead to serious trouble of the urinary apparatus; or it may spread either rapidly or insidiously along the genital canal, beginning with a vaginitis, which may be only very subacute in type; then endocervicitis, endometritis, and salpingitis may ensue, with perhaps the formation of a hydro- or pyosalpinx, and not infrequently acute pelvic peritonitis may supervene.

Occasionally all these complications may be present at some period of the progress of the case. Thus, I saw recently, with Dr. Wakefield, a young lady who had in November, 1892, unfortunately contracted a gonorrhœal vulvitis, followed rapidly by vaginitis and the formation of abscesses in the glands of Bartholini. In September, 1893, she had acute pelvic inflammation and some bladder irritation. In May, 1894, when I saw her, there was considerable pelvic tenderness, the uterus was pushed over to the right by a swollen left Fallopian tube, and was itself extremely tender and enlarged, and semifixed, with a little induration in the pouch of Douglas. Some of the right gland of Bartholini had escaped destruction by the previous suppuration, and was causing great pain. The urethra was secreting pus, and was very swollen and tender and the bladder was tender and functionally somewhat inert, and urine drawn from it contained numerous pus-cells, etc. It was not till January in this year that she was really well enough to be about again.

In infancy vulvitis is usually purulent, but rarely very acute. As puberty approaches, and during menstrual life, vulvitis is either purulent or follicular, whereas in old age it is more of the erythematous type.

The treatment of vulvitis in infancy and old age may be usually directed to the constitution of the patient and to the vulva *alone*, unless in senile cases the inflammation is due to the irritation from discharges such as arise from a senile vaginitis, or a malignant growth of the uterus.

In children the ordinary causes of vulvitis are cold, dirt, or ascarides, associated with a debilitated state of health, such as struma, tuberculosis, anæmia, or the acute exanthemata. These cases are best treated by antiseptic and soothing lotions, which may conveniently be applied by the child sitting in a bath or basin containing the lotion. A useful lotion is warm milk and water, to each pint of which a teaspoonful of borax and a teaspoonful of liquor plumbi subacetatis have been added. This may be repeated several times daily. The parts should then be well dried, and a little lint or wool, soaked in the following, may be applied on lint or some teased wool, so as to keep the parts separated.

R   Liq. plumbi subacet.   ...   ʒss
      Acidi borici   ...   ...   gr.xvj
      Glycerine...   ...   ...   ʒij
      Lactem recentem   ad   ʒviij
Fiat applicatio.

Occasionally zinc answers better than lead, and, if so, the following can be applied with a piece of

wool or a soft brush, after bathing with a borax lotion :—

> ℞ Calaminæ ... ... ℥iss
> Olei amygdalæ ... ... ℥ij
> Aquam calcis ... ad ℥viij
> Fiat lotio.

In every case of infantile vulvitis it is advisable to treat the patient constitutionally also, trying to build her up. Iodide of iron and cod-liver oil are most useful, after a short course of alkalies, vegetable tonics, and purgatives.

In very chronic cases, or where there is much discharge of an irritating character, it may be advisable, after thoroughly cleansing, to apply sol. argent. nitrat. (gr.xx to oz.) over the inflamed area, and then apply a slightly astringent ointment, such as—

> ℞ Ext. hamamelidis ... ℥ss
> Vaselini ... ... ad ℥j
> Fiat unguentum.

Senile vulvitis. Much the same sort of treatment is useful in senile cases due to constitutional causes, such as diabetes, gout, or alcoholism, or to local causes, such as irritating vaginal, uterine, or urinary discharges, or to presence of pediculi, or other less evident parasites.

In these senile cases there are some measures which are especially useful, one or two of which are referred to under the heading "pruritus," for the vulvitis is usually very chronic, and is almost always associated with very acid urine (except in cystitis), or with some form of vulvar rash, especially eczema, and pruritus is a common symptom.

It is advisable, therefore, to give alkalies freely, especially in effervescent form, and to diet the patient by the exclusion of alcoholic, starchy, and saccharine food, and to use antiseptics locally, borax being again a very useful agent.

Sitting in warm water, as directed for infants, is very soothing, and a lotion of lead or zinc (such as the following, or the following ointment) will be found useful, with suitable additions, if pruritus be a marked symptom.

> ℞ Liq. plumbi subacet. ... ℥ss
> Acidi borici ... ... gr.xvj
> Eau de cologne ... ... ℥ss
> Liq. hamamelidis ... ℥ij
> Lactem recentem ad ℥viij
> Fiat applicatio.

Or, ℞ Pulv. calaminæ lævig. ... ℥ij
> Pulv. zinci oxydi ... ... ℥j
> Glycerini ... ... ℥ij
> Liq. hamamelidis ... ℥ss
> Aquam rosæ ... ad ℥viij
> Fiat lotio.

Or, ℞ Liq. carbonis detergens ... ℳxx
> Plumbi acetatis ... ... gr.xx
> Vaselinum album ad ℥j
> Fiat unguentum.

Where vulvar irritation is due to parasites, the following ointment usually gives very rapid results :—

> ℞ Hydrarg. ammoniati ... gr.xxx
> Zinci oxydi ... ... gr.xx
> Plumbi acetatis ... ... gr.x
> Adipis benzoatis ... ... ℥j
> Fiat unguentum.

Vulvitis during menstrual life. Local causes of vulvitis during this period of life are mainly gonorrhœa, irritating discharges from the vagina, local chills, or secondary to acute specific fevers, such as measles or scarlatina.

If the vulvitis be purely localized to the vulva, there is no need to adopt other measures than have been already indicated for the vulvitis of infancy or old age, but if the vagina be primarily or secondarily affected it must be treated simultaneously.

In adult simple uncomplicated vulvitis, baths and sedative applications of warm lead and milk, or lead and starch or barley water, or borax solution, are very useful in the early stages, and liq. opii sedativ (℥j to each pint) may be added, if there be much pain. These should be followed by free irrigation of the vagina with a warm antiseptic lotion. The vagina may in a day or two be lightly tamponned through a Sim's speculum with iodoform gauze to keep the parts separate and permit drainage, and if necessary the whole vaginal and vulvar mucous surfaces may be painted through a Fergusson's speculum with sol. argent. nit. (℥j to ℥j), as fresh surfaces of mucous membrane come into sight whilst the speculum is being withdrawn. Vaginitis does not, however, come into the scope of this lecture.

Follicular vulvitis is an inflammation of the contained sebaceous and mucous follicles, which swell up, their ducts getting blocked, often without much evidence of inflammation of the intervening mucous membrane, except small zones immediately around the follicles.

It is almost always accompanied by, if not caused by, a state of blood at once poor and impure, and requires, therefore, vigorous constitutional treatment, purgatives and iron being particularly indicated.

Local, warm antiseptic fomentations of corrosive sublimate solution (1 per 4000), or sanitas solution (1 in 20) should be used, and poultices avoided. Any suppurating follicles should be opened, and the cavity touched with glycerine of carbolic acid, and it is useful to have the surrounding indurations painted with tincture of iodine daily for two or three days. In chronic cases, or to prevent recurrence, the best application is an ichthyol ointment (5 to 10 per cent.), ichthyol being particularly soothing when mixed with lanoline. A 25 per cent. ointment is often too stimulating. The objection to it is that it has a strong odour and a brown colour.

Inflammation of Bartholini's duct or gland. As a result of either of these, the duct gets blocked, and a retention cyst results, which varies from the size of a small marble to that of an egg. If it be a simple retention cyst, far the best way to treat it is to first try whether hot baths and ichthyol ointment will reopen the duct, and, failing that, to make an incision on the mucous aspect of the swelling, and shell out the cyst entire, applying pressure both from the vagina by means of a vaginal tampon, and from the perineum by means of a pad and T-bandage. The cavity will then usually heal by first intention. If suppuration have occurred, the best, and, in the end, the quickest way, is to follow the same plan, though operation is more difficult, owing to adhesions being present all round the cyst. When the cyst has been removed it is advisable to try to draw the resulting cavity together by means of some silkworm gut sutures passed deeply around the cavity by means of a perineal or a Hagedorn's needle, and then pressure applied as before stated.

Urethritis. Where extension along the urethra occurs it should be gently dilated with bougies, and pure carbolic acid applied through a Bryant's speculum to any painful or red or bleeding area, and if Skene's ducts be swollen, and are protruding through the urethral orifice, a piece of silkworm gut may be passed down, dipped in the same agent, or the duct may be slit up, as one deals with a rectal fistula.

If there be evidence that the bladder is becoming involved, the urethra should be partially dilated, and daily washing out of the bladder should be practised, one of the best lotions being a weak sol. of borax, gr.x or xx to the pint, and the urine should be kept slightly alkaline and antiseptic by a powder of potass. citrat. gr.xx and salol gr.v to x. This latter drug, which changes into salicylic acid and carbolic acid during its digestion, turns the urine brown (carboluria), and as soon as this colour threatens a dose or two should be omitted.

Syphilis. Very little need be said about this, as the manifestations of syphilis vary but little on the vulva from those elsewhere. A chancre on the vulva is readily seen, and easily treated. Secondary rashes as condylomata may occur anywhere both on or around the mucous orifices, and at a later date ulcerative and hypertrophic lesions may be found.

Owing to the fact that a primary lesion may not have been observed, having perhaps been formed on the vaginal walls or the cervix, condylomata are often the first evidence of syphilis. They become multiple by infection of adjacent surfaces, and are capable of infecting another individual, and of then producing a primary chancre.

The treatment is perfect cleanliness, with the local application of lotio nigra, or iodoform paste, whilst the constitutional condition is being treated internally.

Warts. These are usually the result of gonorrhœa, but are sometimes due to syphilis, and now and again seem to be independent of both conditions.

In their treatment, any irritating discharges should be removed by appropriate remedies, perfect cleanliness ensured, and arsenic should be given with the other remedies. If need be, the warts should be snipped off with curved scissors, and their bases touched with acid nitrate of mercury.

Varicocele of the vulva. This is common during the later months of pregnancy, both above the level of the clitoris, and in the labia majora, in the position of Kobelt's bulb. Sometimes a varicocele persists after pregnancy, especially on the left side in constipated women. In all cases the pelvis should be searched for pelvic tumours, the bowels well regulated, straining forbidden, and all tight bands avoided round the waist or pelvis. Diet should be simple, and, if required, a pad may be worn to exert equal pressure upon the varix.

Such varicose veins may rupture and lead to

the formation of an hæmatoma—an effusion of blood into the surrounding connective tissue.

If a hæmatoma obstruct labour, it may, if recent, be aspirated and pressure applied, or if the blood be already semi-solid and the obstruction be great, the tumour must be incised by Paquelin's cautery, the clot turned out, bleeding points secured, and pressure applied with antiseptic gauze, etc., etc. As a rule, however, no surgical interference is required in cases occurring apart from labour, unless suppuration supervene, when the tumour must be treated as an abscess, opened and drained.

Hernia. Intestine or omentum may descend through the inguinal canal along the round ligament, and has to be treated like herniæ elsewhere. In the same position, running downwards from the external abdominal ring into the labium majus, may be found a swelling due to distension of the unobliterated canal of Nuck by fluid. This may be encysted and be irreducible, or may be continuous with the peritoneal cavity. If the fluid be encysted (hydrocele) the cyst may be aspirated, and if it refill, it should be again aspirated, and its cavity injected with tr. iodi.

Sometimes an ovary may be present in the inguinal canal, and be found as a tender swelling on the front of the pubic symphysis, and during married life is a cause of much pain. If this be an acquired hernia it may, perhaps, be reduced, and a truss applied, or, if irreducible, it may be protected from pressure by a concave pad, but if it become inflamed its removal is demanded.

Sometimes a swelling is found which exactly corresponds in shape and size to a hernia of the ovary, but on removal is found to be a testis, the case being one of internal male organs, and external female organs (transverse hermaphrodite).

Malignant Disease. Epithelioma of the vulva is the commonest form of malignant disease, though scirrhus is not unknown.

The growth has to be removed by the actual or Paquelin's cautery, as the bleeding is sometimes very profuse.

The slough may then be dressed with a solution of pepsin and carbolic acid, and allowed to granulate over.

Adhesions. In addition to imperforate hymen the vulva may become adherent behind the hymen, as a result of some previous inflammation, dating, perhaps, from infancy. This may unite the two labia majora, arising posteriorly from the bottom of the fossa navicularis, or, more commonly, a thin membrane is found which is continuous with the posterior fourchette, or anterior margin of the perineum up to the level of the urethral orifice. Such a condition may be congenital.

These membranous diaphragms are easily torn down the central line by the fingers or the pressure of a probe, but sometimes are apt to grow up again, and if this occur, spite of attempts to keep the parts separated, a small ring of silkworm gut may be tied at the normal level of the posterior fourchette.

Occasionally the clitoris has its prepuce adherent, and much local irritation is caused by retained smegma. This also may be congenital. The prepuce easily separates along a central raphé, and, with care on the part of the nurse, does not reunite. I have seen this occur, congenitally, in three children, whose brother had had to be circumcised for congenital phimosis.

Vaginismus. This distressing condition may be the result of a pure neurosis or may be produced by intra-pelvic causes, or as a sequence of failure of the marital act on the part of the husband. Very frequently, however, the cause is purely local, due to cracks or fissures or ulcers about the vaginal orifice, the hymen, the fossa navicularis, or the ducts of Bartholini. Such conditions must be appropriately treated, and, if the habit of spasmodic contraction still persist, the vulva must either be gradually stretched and increasing sizes of vaginal "rests" worn, or forcible dilatation may be performed under anæsthesia, and a large "rest' worn for a few hours daily for some weeks.

Sometimes when the hymen is very sensitive, and the least contact with it causes an irrepressible contraction with pain, it is necessary to cut it entirely away with curved scissors. It may also be desirable to cut, submucously, with a tenotomy knife, any muscular fibres which are mainly at fault (noted when the patient is coming round from the anæsthetic); but, as a rule, forcible dilatation suffices.

Kraurosis Vulvæ. In my last lecture I described this peculiar state, occurring usually after the menopause, its most prominent signs being a glistening white appearance of the inside of the labia, with here and there a spot of ulceration, and a tendency for atrophy and shrinking of the parts to soon follow. Soreness, pruritus, dyspareunia, are the chief symptoms.

It is best treated at first by a half-strength unguent. hydrarg., with the addition of some

suitable agent for co-existing ulceration or pruritus. Great improvement follows this, and if it be used freely a cure will temporarily, at all events, ensue. I have seen this happen twice. In one case, that of a midwife, an ointment of

R	Unguent. hydrarg.	...	ʒj
	Unguent. plumbi	...	ʒj
	Olei menth. pip. ...	...	ʒj

Fiat unguent.

had been applied, advantageously, in small quantities twice daily for two months, and the patient, being dissatisfied with the slowness of the cure, determined to hurry matters by rubbing in the ointment several times a day. In ten days she was salivated and lost several teeth, but the vulva was free from all discomfort, and looked quite pink and healthy and supple. In six weeks, however, it began to recur, and was with difficulty kept under control.

In cases where much suffering is caused the mucous membrane may be dissected off; but I have found very good results from scraping with a sharp pronged fork (shown), and subsequent weak mercurial and lead inunction. At the same time the contracted vaginal orifice may be opened up by incisions.

Pruritus pudendi. The last condition to which I shall refer to-day is pruritus—a most distressing *symptom* of many very different diseases. It may be a pure neurosis, especially at the climacteric. It may be "reflex," as in some women during menstruation, or in pregnancy, where it may take the place of the reflex vomiting, or in cases of *early* carcinoma cervicis, or of mucous polypus, or of prolapsed ovary. It may accompany rashes of all sorts, especially eczema, be due to dirt, ascarides, pediculi, or other less evident parasites, to vascular urethral caruncle, to vulvar glandular swellings, to irritating vaginal discharges as in advanced cancer of uterus, or to irritating urine as in vesico-vaginal fistula, chronic cystitis, very acid or gouty or diabetic urine. Sometimes a condition which may best be described as a local seborrhœa is found.

The treatment is as varied as the causation, and palliative remedies are not very satisfactory, nor may they continue to give relief even if they act like a charm at first. About the most certain remedy is a teaspoonful of the following powder in a pint of warm water :—

| R | Sodii biboratis ... | ... | ʒiij |
| | Ol. menth. pip. ... | ... | ʒij |

Fiat pulv.

If this is not liked it is because, in some cases, it produces a feeling of coldness and numbness, which is not appreciated.

Other local applications which are useful whilst the cause is being removed are the following :—

Cocaine ointment, gr.viij to ʒj.

One of the lead or zinc lotions already given, with acid. hydrocyanic (ʒss to ʒviij).

Menthol ʒj, Olive oil to ʒj.

Or, Aqua menth. pip. (warmed).

In senile pruritus without obvious origin, corrosive sublimate solution (1 in 2000) affords rapid relief, apparently by destroying some parasites not discoverable, and it is a good plan to give salicylate of soda internally (gr.x every four hours).

In diabetic or gouty pruritus, after thorough cleansing, an ointment of salicylic acid gr.xx–ʒj may be well rubbed in, or the well-known Lassar's paste :—

R	Acidi salicylici...	...	gr.xx
	Zinci oxydi		
	Pulv. amyli ...	āā	ʒiv
	Vaselini alb. ...	...	ʒij

Fiat pasta.

In all forms of pruritus, warm baths are soothing, one of the best being equal parts of milk and barley water, with borax (ʒj to a pint); or liq. carbonis detergens and water (ʒj to the gallon).

If the pruritus be reflex or neurotic, valerian, valerianate of zinc, and the bromides are useful, and a course of baths at a suitable health resort in cases of gout and acidity will hasten the cure.

As a last word, may I emphasize the necessity of treating most vulvar diseases from the constitutional as well as from the local standpoint. Like any other mucous orifice of the body, it is often affected by conditions which are, as we have seen, solely or mainly the results of constitutional states, and in any case, even if the vulvar trouble be purely local at first, the general health should never be overlooked.

Intestinal Antiseptic.—Paraform, according to Aronsohn, is a very strong intestinal antiseptic, superior to B-naphthol, iodoform, salol, dermatol and benzonaphthol, and having a marked inhibitory action on the propagation of bacilli. Paraform is a solid, white crystalline polymer, produced by heating formaldehyde in watery solution. One grain is said to be capable of completely sterilizing 200 grammes of urine.

(*Jour. der Pharm. v. Elsass-Loth.*)

THE

PHYSICAL CULTURE OF YOUTH.

A Paper read before the Gloucestershire Branch of the
British Medical Association at a Meeting held at
Cheltenham, Dec, 18, 1894,

O. FOWLER, Esq., of Cirencester, President, in the Chair.

By NOBLE SMITH, F.R.C.S. Ed.,

Surgeon to the City Orthopædic Hospital, and Surgeon
to All Saints' Children's Hospital, London.

IN the prevention of deformity in youth, or in
other words, in the cultivation of the stature during
growth, there are two chief lines of action to be
followed, the one being the adoption of all methods
which tend to develop the bodily form into a
healthy, upright, and physically strong condition,
and the other the avoidance of all counteracting
influences.

There is one great principle in the physics of
the human body which it is well to recognize at
the outset, and that is the fact that long continu-
ance of a bad position, even to a slight extent, is
far more harmful than interrupted positions of
more severe character; in fact, it matters not what
postures the individual may assume for very short
periods, and in childhood nothing is better for the
health than natural and almost constant movement
in all directions.

The most potent obstacle to the proper develop-
ment of the child is, I consider, the clothes he
wears. This is not at all a necessary evil, but, as
a fact, the ordinary construction of clothes is
absolutely wrong in the great majority of instances,
and I think I am well within the mark when I
state that with at least 90 per cent. of children,
including all classes, the evil I refer to exists.
Many of these errors in construction of clothes are
the outcome of views entertained by us of the
medical profession, views which, although sound
in theory, have become absolutely wrong in
practice.

To begin with: We have always opposed the
tight constriction of any part of the body; conse-
quently, we have objected to tight bands round
the waist, and the suspension, especially in women,
of heavy clothes from the pelvis; also we have
protested against tight garters round the leg—the
former because they interfere with the organs of
the pelvis, and the latter because they retard the
circulation and give rise to or exaggerate a tendency

to varicose veins. These objections, as I have
already said, are founded upon sound theories, but
what results have they given rise to? So much is
there a fear of constricting a child, either in the
waist or legs, that the clothing has been almost
entirely suspended from the shoulders, giving rise
to incalculably more harm than ever could arise
from the former practice.

Children, especially girls, have to wear stockings,
and to keep these stockings up an elastic suspender
is attached, this being fastened either to a light
stiff jacket or a corset which is kept up by shoulder
straps, or else the suspenders are carried direct by
straps over the shoulders. Then other articles of
clothing are also kept up, more or less, by shoulder
straps, and these straps are not placed close to the
neck, where they would be less harmful, but to
the apices of the shoulders, where they constantly
tend to bear the latter downwards and forwards,
giving rise to stooping shoulders and to a poking
head, depressing the chest, and acting as a constant
source of irritation to the wearer.

It is hardly necessary for me to point out the
evils of this practice, how a posture thus engen-
dered lessens the vital capacity, and so affects the
physique and health of the body; how it favours
the liability to coughs and colds, and even to
phthisis; how the bending forwards of the spine,
and the efforts, slight but constant, to hitch up the
shoulders or to overcome this dead weight may
be the starting-point of curvatures of the spine,
and as girls are subjected to a greater extent to
this evil clothing, how this is probably one cause
of the greater prevalence of spinal curvature in
their sex.

Clothes must be fastened somewhere, and it is
desirable, I maintain, to divide as far as possible
the points of attachment. The clothes which
surround the trunk may be kept up by their attach-
ment over the shoulder, but this should be close
to the neck, as takes place with the ordinary
flannel vest. No part is more fitted for the sus-
pension of the nether garments than the hips, and
this fact is well known to all of us. Every man
who has to work his arms takes care to get rid of
all encumbrance about the shoulders. The sailor,
the stableman, or any other worker gets rid of his
braces and uses a belt, the natural indication being
to leave the shoulders free for action.

If, for keeping up the stockings, suspenders are
used, they should be fastened to the waist (but
even when fastened here they may bend the body

forwards from being too tight), and not to the garment which eventually takes its bearings from the shoulders. Garters may be used without a fear of evil, certainly with children, for they need not be so tight as to interfere with the circulation. It is only in exceptional cases, and chiefly in adults, that garters under any circumstances become harmful.

A well-developed chest, with straight back and well-placed shoulders are an exception in civilized countries, chiefly, I believe, as a consequence of the evils I have pointed out. Children grow up with ill-developed chests and stooping figures, and with these we have frequently to deal. Even severe drilling in later years never entirely overcomes the harm thus done in youth. These results are commonly attributed to school postures, and doubtless these postures are bad enough ; but one reason why they produce so much havoc is, that the bad figures have been first of all engendered by the wearing of improper clothing.

In dealing with these children one always finds that their clothing has been fitted from a dressmaker's or a tailor's point of view, *but in the bad positions*, and any attempt to hold them upright and bring back the shoulders into a proper place is counteracted at once by the tightness of the garment across the chest. The bad influence of this wrongly-shaped clothing is commenced long before the child begins scholastic occupation ; but when we come to the time of life when school commences, we are then met with difficulties against which it is almost hopeless to contend.

FOOD. Another very important point is the nourishment of growing children. It is a serious accusation to make, and one which I should not venture upon had I not a very considerable amount of evidence to bear me out, that there are very few schools in this country where the children are sufficiently well fed to enable them to grow up strongly, let alone to meet the extraordinary strain upon their efforts of development caused by the immense amount of mental work which is thrust upon them. A lack of nitrogenous diet, a lack of care and niceness in cooking, a monotonous character of food which damps the child's appetite, all combine to bring about this bad effect.

One of the few members of our profession who has boldly stood up as a champion in this matter is Mr. Henry Power of St. Bartholomew's Hospital, and I greatly regret that his paper upon this subject

has not been published. The four chief points in which school diet fails are :—

1. Insufficient quantity of nitrogenous food.
2. Indifferent quality.
3. Indifferent cooking.
4. The long periods between solid meals.

1. That the quantity is insufficient is proved by the great number of feeble young people who are turned out from these institutions, and the fact of the large number of delicate girls and boys who come under treatment for conditions which clearly indicate the want of nitrogenous strength.

2. I might assume that the indifferent quality is a truth well known to most of us, but I may here mention a fact which lately came before me, that at one of our largest public schools for boys the meat had to be sent away from the table because it was absolutely bad several times during one term.

3. Indifferent cooking is very prevalent in schools, and the general view that the complaints of young people are not worth attention, leads to this matter being overlooked.

4. The time between mid-day dinner and next morning's breakfast is seldom interrupted by any solid meal of meat, for even when meat is placed upon the table its unattractive condition repulses the appetite and it is frequently not touched.

The story of these deficiencies as regards the food of young people is probably an answer to the following questions : First, Why is it that so many children grow up with delicate constitutions ? Second, Why is it that so many thousands develop weak and crooked spines ? Third, Why is it that tubercular disease makes so much havoc in this country ?

The alteration of children's clothing can be made comparatively easily, when the evil is fully recognized. The change in diet may be a matter of greater difficulty ; but there are other matters for consideration when we have to deal with patients who are suffering from weak and crooked spines, or who are in any other similar way affected by physical disability.

At the time when they come before us for treatment the mischief has probably, I may say almost certainly, been developing for several years, a development which may have been quite unnoticed, or noticed only as a tendency to stoop, and to poke the head forwards. The delicate structures involved have been gradually giving way into un-

natural forms. The vertebræ have by degrees assumed wrong shapes, and the ligaments which hold them together have become relaxed, producing a total inability on the part of the child to hold the spine in an upright position for any lengthened period.

It is almost a universal idea that muscular weakness is the cause of this condition, and although in many cases the muscular system may partake of the general debility, I would urge that the muscular system is by no means always at fault, and even when it is, such weakness is a very small matter in comparison with the commencing bony deformity and relaxation of ligamentary structures.

The muscular weakness can be easily dealt with at any time of life, and a few weeks' training will generally get the muscles into a strong and healthy condition, but this treatment will not restore the misshapen bones. If the shape of the bones of the spine have been modified by months or years of stooping, the temporary improvement in position during the necessarily short periods during which the exercises can be maintained (at each time of exercise) is quite insufficient to produce any permanently good effect. It is only by continued maintenance of the spine in an upright position that absolute change in its form to a normal shape can be effected.

I am not decrying exercises, for I firmly believe in their efficacy and advantages when made use of at a proper time, and for a proper purpose, but I would urge most strenuously that they are not the best, or even necessary means, of correcting the malposition I am discussing. I would even go further and say, that a great many of the quickly-grown delicate children and young people to whom I refer are the better for a period of rest rather than an excess of exercise, and that they will gain strength and health much more quickly by moderate repose than by excess of drilling. Keep their spines from falling into a crooked position sufficiently long to allow the bones and ligaments to recover their natural shape and strength, and then adopt a system of exercises to maintain the improved position you have produced.

When I talk of repose I do not refer to the forced and irksome recumbency so often recommended, but simply to an avoidance of excess of exercise, of all exercise which produces fatigue, long monotonous walks being especially harmful.

These cases of weak spine have a great likeness to those of a quick-growing plant which a careful gardener knows will fail to develop into a strong growth, unless he trains it up with the help of artificial support ; in fact, the whole subject is one of cultivation *versus* neglect.

The evil of prolonged physical exercise in young people has been wisely pointed out by Dr. Lauder Brunton in his very learned and logical Harveian Oration at the Royal College of Physicians, and those interested in this matter would do well to refer to the report of this lecture as given in the Medical Journals at the time.

If we lived absolutely natural lives we should probably grow up straight, but then there would be no misfitting clothes, no school, and no sitting on chairs. I would ask, why do we sit down? and the natural answer would be "to rest." Then I would further ask, Why, if we want to rest every other part of the body, do we not want to rest the back? It is quite exceptional for the back of the sitter to be supported by the back of the chair.

I think it highly probable, if not certain, that if children were obliged or allowed to thoroughly rest their backs whenever they sat down, we should have very few cases of weak and curved spines. However, we have to deal with the results of the evil postures, and however necessary or desirable it may be to prevent deformity, we are far more often called upon to deal with a condition of prolonged weakness than to suggest methods of prevention. What is to be done? If you agree with me that exercises under these conditions are not effectual in removing them, but that prolonged maintenance of the spine in a correct position is necessary to allow of a restoration of the bones to a proper shape and a return of firmness to the ligaments, we must then consider how these latter requirements are to be attained. We have recumbency in a supine or, better still, in a prone position; but let us consider what this will involve. It is an irksome proceeding. It debars the patient from even moderate exercise, it engenders a condition of invalidism, and, above all, it is not satisfactorily effectual; for whenever the patient moves from the recumbent position and returns if only for a short half-hour to the bad position, the effect of the interruption is potent in undoing the good which hours of rest may have produced. What I would wish to do is to keep the spine always in a straight position until it has regained its proper

shape, not interfering with ordinary movements, not enforcing irksome restraint, but preventing the spine from getting into bad positions, and this without interfering with development and without interfering with the action of the muscles.

CLINICAL
DEMONSTRATION OF CASES.

Given at the Monthly Meeting of the North-West London Clinical Society, held at the North-West London Hospital on Wednesday, Feb. 13, 1895.

MAYO COLLIER, Esq., F.R.C.S.,

In the Chair.

DR. KNOWSLEY SIBLEY showed the suprarenal capsules from a case of Addison's disease, which had died in the hospital three days previously. The patient, a girl aged 20, was shown at a meeting of the Society* two months previously as a case of somewhat anomalous pigmentation, especially of the face, neck, and hands, without many symptoms beyond a rather rapid pulse. Since that time the pigmentation had considerably increased, vomiting came on, and the patient became markedly weaker. She was admitted into the hospital in the end of January. After the first two or three days' rest in bed the pulse quieted down from about 120 to 90. On February 1st vomiting set in. On February 5th the vomiting became much worse and could not be relieved by treatment. The pulse became rapid and very feeble. February 7th, the patient lay in a state of extreme exhaustion, vomiting on the slightest movement, and during the night she died suddenly. At the *post mortem* the body was well nourished, all the organs appeared healthy except the suprarenal capsules, both of which were much enlarged, somewhat adherent to the surrounding structures, very hard and nodular. On section there was no sign of soft caseous tissue.

DR. CAMPBELL said that, in common with many of the other members, he had seen the case at a former meeting. Pigmentation was then so little marked that doubt existed as to the nature of the case. Within a short time, however, pigment was deposited so rapidly and universally as to produce a wonderful change in the girl's appearance. Before death she was at least as dark as a mulatto. Another point was the apparent absence of severe anæmia. He did not know whether the corpuscles had been estimated, but to judge from the colour of the lips the blood was but little impaired. This case taught the important lesson that death in Addison's disease might occur suddenly and without very definite warning.

DR. GUTHRIE observed that in this disease there seemed to be no relation between the amount of pigmentation and the gravity of the case. Neither does a high degree of pigmentation point necessarily to Addison's disease. One of the most extreme instances that had come under his observation had been in a case of Graves' disease, where *post mortem* the adrenals were found to be quite healthy. He had taken this case, when first exhibited, to be one of Graves' disease, on account of the acceleration of the heart's action. Marked pigmentation was sometimes found in chronic Bright's disease, and Dr. Guthrie referred to two cases which, before death, had been thought to be Addison's disease, but which *post mortem* showed only kidney changes. Referring to the pathology of Addison's disease,

* *Clinical Journal*, vol. v., No. 10.

it was strange that only tubercular degeneration of the adrenals seemed to cause the symptoms ; and the similarity in some respects between Graves' and Addison's disease suggested the belief that derangement of the sympathetic had much to do with both.

DR. GILL referred to cases within his experience where caseous degeneration of the suprarenal bodies was found *post mortem*, although the symptoms and mode of death in no way pointed to Addison's disease.

DR. STOWERS exhibited a case of lupus erythematosus on the face and right ear of a girl, aged 18 years. The disease commenced two years since over the right malar bone as a circular patch about one inch in diameter. Linear scarification, assisted by the application of a 25 per cent. solution of carbolic acid, completely removed it, leaving so smooth and superficial a scar as to be almost unobservable. Three months ago several new patches developed on the right cheek and ear, and one over the left malar bone, which will be treated in a similar way. The difficulties of diagnosis in the early stage of this disease were pointed out, and the operation of linear scarification described in detail.

DR. STOWERS also exhibited a woman, aged 35 years, suffering a rare form of tertiary dermato-syphilis. The disease had existed upwards of three years, and was remarkable on account of the regions involved (face, ears, and flexures of elbows), and the characters it possessed in common with chronic eczema and erythematous lupus. The diagnosis was confirmed by the ordinary concomitants of syphilis, including the fauces and larynx, and also by typical ulceration on the anterior surface of each leg. The patient was improving rapidly under treatment.

MR. JACKSON CLARKE said that he considered the name of lupus bestowed upon these cases as very unfortunate. They had little in common with lupus, although there was the same tendency to the permanent destruction of tissue. He had carefully investigated the microscopic character of the patches in this disease for Mr. Malcolm Morris, and had found no trace of tubercle. Neither were the ravages attributed to it always so great as was supposed. He mentioned cases in which the only present appearances were those of a raised border with dilated orifices, blocked with sebum at the circumference, about one or two of a group of scars. He had once seen a patch of erythematous lupus on the conjunctiva. The ordinary methods of treatment aimed at a cure by anticipating the natural process. He asked whether Dr. Stowers had used pyrogallic acid as a local application.

MR. MAYO COLLIER concurred that the name lupus erythematosus was misleading. The process left a scar, and in that way only resembled lupus. He referred to the benefit to be had from quinine in this disease.

DR. WILBE raised the question of the administration of thyroid extract in lupus erythematosus.

DR. STOWERS in reply to Mr. Clarke, said that pyrogallic acid often caused extensive inflammation of the skin, and it was desirable to employ a remedy more under control. Scarification, supplemented by carbolic acid lotions, provided such a remedy. Lupus erythematosus sometimes cured spontaneously. He agreed with Mr. Collier that quinine was useful, but in cases where the disease was spreading rapidly, scarification should be resorted to. He had known of thyroid extract being used, but was not aware of any good results so obtained.

DR. HARRY CAMPBELL showed the heart of a man, æt. 66, who had died from simple aortic obstruction, an occurrence so rare, he remarked, that death from this cause had actually been denied. The aortic orifice was converted into a narrow chink, barely admitting the blade of a scalpel, while the valves were perfectly competent. There was enormous hypertrophy and some dilatation of the left ventricle, the mitral valves being competent. Clinically the most notable features were (1) the maintenance of good arterial tension till within a few days of the patient's death ; (2) the engorgement of the lungs and of the systemic veins, although the

mitral valves were competent; (3) the almost complete imperceptibility of the apex beat, in spite of the great hypertrophy. Dr. Campbell attributed this latter circumstance partly to the existence of some degree of emphysema, but chiefly to the inability of the left ventricle to rapidly expel its contents.

DR. GILL related the history of two cases of aortic stenosis occurring in the same family. In the first the post-mortem examination showed a very extreme degree of obstruction, and this had been the cause of death. The other patient had recovered from an attack of acute cardiac trouble, and died some years later, having in the interval been the subject of chronic bronchitis. In neither case was there evidence of regurgitation.

MR. MAYO COLLIER showed a girl, aged 12, with talipes equinus, the result of infantile paralysis.

The deformity was of three years' duration. The condition of things now was that the posterior muscles had shortened and contracted.

The anterior muscles were not paralysed, as evidenced by the marked efforts to bring the foot to a right angle—the muscles had recovered to a large extent, but were unable to overcome the contracted and powerful posterior set.

The nutrition of the limb was not much impaired.

These cases were often improperly subjected to galvanism and massage in this stage.

Until the strain was removed from the anterior set by dividing the tendon of Achilles, galvanism, friction, and boots were useless. On approximating the origins and insertions of the anterior set, these muscles would regain their lost elasticity; the intermuscular vascular plexuses would dilate, and injurious pressure would be removed from the nerve plates. This in many cases was all that was required to restore complete volitional control and locomotion to the limb. Without the preliminary step of giving the weakened muscles physiological rest by removing the antagonism of their opponents, galvanism, massage and injections of strychnine were useless.

MR. MAYO COLLIER also showed a boy, aged 13, with flat-foot of so marked a character as to prevent locomotion. These cases were not rare, and no doubt those present would wonder at such a case being exhibited. Mr. Collier's object was to make a few remarks on the causation and treatment of flat-foot. There was no tendency to a displacement forwards and downwards of the astragalus when the normal foot rested on a horizontal surface.

The astragalus rested securely and without any extraneous support on the os calcis when that bone was held in its normal position.

Even in the dried tarsus without any ligamentous or other connections, the astragalus rested securely on the os calcis, and could not be displaced forwards by any force vertically applied.

On tilting the os calcis by raising the heel the astragalus slid forwards, downwards, and inwards, and so in the recent state pressed unduly on the calcaneo-scaphoid ligaments and other structures in this situation, and led to elongation of the tarsus and flat-foot.

This simple demonstration afforded the most valuable clue to the proper treatment of these cases, namely, to, first and foremost, restore to the foot the normal position of the os calcis by removing all heel to the boot.

Without this primary and essential step all pads, tonics, exercises, and galvanisms were comparatively useless.

In very many cases the entire removal of the heel from the boot was sufficient to restore locomotion and comfort to the affected limb.

DR. CAGNEY, commenting upon the first of Mr. Collier's cases, agreed that in such cases, namely, where contracture had been allowed to develop and much valuable time had already been lost, a preliminary to active treatment should be a relaxation of the wasted muscles by tenotomy of their opponents. Inasmuch, however, as Mr. Collier's remarks seemed likely to convey the impression that nothing further

was needed, and that other treatment was superfluous, he felt constrained to protest against this doctrine—a doctrine which represented the average feeling of medicine—much more than the average of surgical opinion. Nevertheless, it was this masterly inactivity more than anything else which filled the land with cripples. He maintained that there were constantly coming to every hospital children who had had infantile paralysis—in whom an indispensable limb was useless for want of a little added strength, a little further bulk of muscular tissue, which would suffice to move the joint. He was not advocating one special method of treatment, but he would refer to the researches of Mr. Bland Sutton and others as proving that even in health, and especially in young people, there were present in the fasciæ immature muscular fibres, and to work these up in paralyzed children no effort should be spared. He could conceive of no work more worthy of the care and energy of the physician. It was to be accomplished in many ways. Massage and electrification—not necessarily galvanism—were the most obvious expedients; but, in addition, there were passive movements, the application of warmth, the hot air bath, heat-forming foods, intramuscular injections of strychnine, and a host of remedies coming under the head of rational therapeutics. He believed that he had never seen a case of infantile paralysis which was not susceptible of benefit, and very many could be made with care to turn the scale from inefficiency to a useful vitality.

MR. JACKSON CLARKE observed that in combination with other measures he had found regular gymnastic exercises of great service in flat-foot, whilst in cases of hallux rigidus exercises had no good effect.

REVIEW.

The Aseptic Treatment of Wounds. By Dr. C. SCHIMMELBUSCH. Translated by Dr. A. T. RAKE. (Lewis.)

Published at 5s.

This is an exceedingly interesting little book of 211 pages, giving many practical details on the aseptic management of wounds, and methods by which instruments and dressings may also be rendered aseptic. In the author's opinion boiling any article in a one per cent. solution of carbonate of sodium ($NA_2 CO_3 10H_2O$) is the best possible method of sterilizing it; especially is this true for steel instruments, which are not rusted by this solution. The apparatus required for aseptic surgery, as described in this book, is rather expensive for general work; but for surgeons who are frequently operating, and for hospital theatres, some such apparatus is essential, and probably is now in use all over England. We know of no other book which gives in such a small compass so much practical information about the now universally desired aseptic operation. A word of praise must be given to the translator, who has done his share of the work in a most admirable manner.

A POST-GRADUATE LECTURE

ON

THE TREATMENT OF MUSCULAR CONTRACTION.

Delivered at the National Hospital for the Paralysed and
Epileptic, Queen's Square,

By W. R. GOWERS, M.D., F.R.S.,

Physician to the Hospital, and Consulting Physician to
University College Hospital.

GENTLEMEN,—You are familiar with the fact that consequences of disease are sometimes more serious than the maladies from which they result. I do not now refer to the cases in which one definite disease is produced by another, such as the paralysis due to the poison that is the result of diphtheria, which causes such grave after-effects. I desire to draw your attention only to some simple results of disease, results that are the necessary consequences of the primary affection, —which we may almost describe as the natural effects of unnatural states. But, although they are normal consequences of abnormal conditions, they reach, by their simple increase, a degree which constitutes so definite a disorder that we are inclined to regard it as a morbid process. A morbid process it is, so far as its effect is concerned, although it is not a morbid process in its nature. It is disease only because it is an excess —because, in the absence of the normal limitation, it passes on to a condition which disorders normal action.

This being the nature of the contractions of which I am about to speak, I must point out to you their causes and mechanism, that you may understand them, and that you may discern their nature when you encounter them; that you may know what they mean, and that you may know how to remove them—to remove them if it may be, to lessen them if they cannot vanish, and, above all, to anticipate their occurrence, to discern the conditions in which they will develop and yet in which they need not develop, to prevent them in the strictest sense of the word—to come before them, to stand in their way, to *obviate* them.

Such a secondary process, arising from the unrestrained action of processes which are normal, is the cause of many of the results of disease which are most difficult to remove. Indeed, removal is only possible when the normal restraint can be renewed. The process of diminution even then is very slow. The renewal of restraint is gradual, and has to act on a tendency that has become far greater than normal, and, moreover, has a tendency to increase. This has first to be arrested before diminution of the abnormal degree can be achieved, and the normal restraint re-established, if its recovery is adequate.

I use these general terms because the conditions described, and the influences referred to of restraint on the one hand, and excess on the other, may be perceived in many morbid states, and their discernment enables us to understand better many processes and many consequences which outlive their causes. They often seem like independent maladies when they are only properly to be regarded as runaway vitality. For they depend really on the normal power of living structures to adapt themselves, by their nutrition, to conditions different in character or only in degree from those of health.

This process, as I say, may be met with in many affections, but in none more clearly than in the condition to which I want to direct your attention to-day, the muscular contraction met with in many paralysing maladies of the spinal cord and of the nerves.

The tonic contraction of muscles that occurs in organic disease may be divided into two chief forms, easily distinguishable, and important in their significance—that which yields to an attempt to extend the muscle, and that which cannot be overcome.

I propose to consider to-day only the form that cannot be overcome. The calf muscles are often thus contracted as I show you in this patient, who is recovering from multiple neuritis. When the foot is pressed up by the hand, placed against the sole, the calf muscles resist, the contraction yields a little, and the foot is brought almost or quite to a right angle with the leg; but however long you continue the pressure the foot goes no further.

That is the contraction which depends upon tissue-changes in the muscles, causing it to be structurally shorter than normal. It is of very great practical importance. You may remember observing it in the boy with idiopathic muscular atrophy whom we examined last week. The first point for us to consider is to what it is due.

It is met with especially in muscles that are less paralyzed than their opponents, and is most common in the calf muscles, which extend the ankle joint, when the flexors in the front of the leg are paralyzed. But we also meet with it when both sets of muscles are paralyzed, and also in cases in which there is no paralysis, provided extension of the foot on the leg has been maintained for a long time. It occurs also in the flexors of the hip and of the knee if, from any cause, the patient lies in bed for a long time with these joints flexed. If you scrutinize the conditions in which it is met with, you will find that it is due especially to posture—that even paralysis is only effectual by inducing a certain posture. You know that there is an extraordinary capacity of the muscles to adapt themselves to posture. Whatever the position of a limb or joint, the muscles must always continue in a certain state of slight gentle tonic contraction—"physiological tonus" or "tone" as it is called. If a limb is moved passively, the muscles which are relaxed do not wrinkle up; they remain contracted in exact proportion to the approximation of their attachments, and the muscles which are extended elongate, but remain in similar tonic contraction in exact proportion to the increased distance between their attachments. That capacity for adaptation to posture is evidently one phase of the condition which physiologists call "tone," and which depends upon the connection of the muscles with the spinal cord. From the nerve cells, in the anterior cornu, motor fibres proceed to the muscle. From the tissue between the muscular fibres, other nerve fibres pass up to the posterior cornu of the cord, and are connected, by a mysterious interlacement of fibrillæ, with the branching processes of the cells of the anterior cornua. On the integrity of that arrangement this adaptation to posture and this muscular tone seem to depend. In consequence of that, if the attachments of a muscle are permanently and constantly approximated in consequence of posture, the changes which, as you know, are always going on in every structure and every tissue of the body, alter the muscular fibres and the interstitial tissue,

in accordance with the diminished length, so that after a time you are unable to extend the muscle—unable, that is, by any attempt which you may make at the time. We meet with this in cases of unequal paralysis, and also in cases in which one posture is constantly adopted. This effect of paralysis may be always traced to its influence in causing a certain position of the parts. That is the great cause of the various forms of talipes that result from the unequal palsy of muscles in infantile paralysis. The most common of these forms, that to which I want specially to draw your attention as typical of the others, is the contraction of the calf muscles when those in front of the leg are paralyzed.

We meet with it also as the result of posture determined by pain, and also of posture determined by simple comfort. It is probable that when there is infantile palsy with this sequel, the contracture of the muscles is facilitated by the fact that they also have suffered a little, that there are slight interstitial changes, nuclear multiplication, with increased development of new fibrous tissue which contracts. But remember it is not a consequence of the palsy; it occurs without it—I mean it is not a consequence of the slight palsy of the contractor muscles; it is the consequence of the persistence, for a long period, of the state that results from the capacity of the muscle for adaptation to posture.
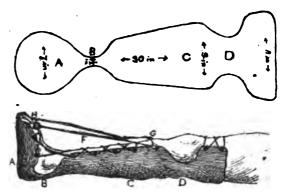
It is a grave inconvenience; it hinders the recovery of use of the legs for standing and walking in patients who have otherwise gained sufficient power. It often needs division of tendons in order to permit the wrong posture to be rectified; but it does not always need it. In many cases, unless there is shortening of the leg, the persistent, long-continued extension involved in the act of walking elongates the contracted muscle to its proper length. And that is the case in all conditions in which perfect recovery of the damaged structures causing paralysis is possible. It is conspicuous in adults—in whom no hindrance to growth can occur. In multiple neuritis, for instance, we have much greater palsy in the muscles in the front of the leg than in the sural muscles. The latter contract, in consequence of the posture permitted by the weakness of the flexors of the ankle. It is a disease which passes away under good conditions; and however great that contraction, it always in time yields to the attempt to stand and walk. Tenotomy is never needed. Tenotomy is only indispensable when the condition

is due to a state from which recovery is impossible. In infantile paralysis it is frequently needed; because infantile spinal paralysis depends upon destruction of the grey matter of the cord; recovery is impossible then, and without return of power in the opponents, the contraction that results cannot be removed.

But that which is difficult to remove may be prevented. The great cause of this contraction of the calf-muscles, permitted by the affection of the muscles in the front of the leg, is the action of gravitation in determining the posture of the foot as the patient lies. The foot "falls," i.e., always sinks into a position of extension. The calf-muscles become shortened, first by active, extensible contraction, but this becomes fixed by nutritional changes, and ultimately is absolute by structural change. But the falling of the foot need not occur. It is a point of great practical importance and is almost entirely neglected. There never need be this shortening of the calf-muscles from loss of power in the anterior tibial muscles. It is only necessary to keep the foot always up, and the contraction cannot occur. It may be kept up either by some support below the sole, such as a plank or a thick sandbag, or else by some traction of the foot. Support from below cannot, however, be maintained in adequate continuity. I have tried every form of support that could be devised and have been compelled to fall back on traction. The force exerted need not be great; a very gentle elastic traction, long continued, will keep the muscles adequately extended, and prevent the occurrence of this most troublesome result. But the contrivance of such traction is a much less simple matter than it may appear. There must be a place from which it is exerted. It must come from some part near the knee. However gentle it is, by long continuance it becomes annoying, then painful, and ultimately unbearable. It can be borne, indeed, for a very little time when there is tenderness, as there is always in the condition in which it is most needed—cases of multiple neuritis. I tried traction from the upper part of the bedstead, but the changing posture of the patient made this useless. At last, after many trials of various forms, an idea occurred to me of a plan by which the traction could be exerted from the upper part of the leg near the knee, which has proved perfectly successful. I described what I desired to our excellent resident medical officer,

Dr. Whiting, who speedily carried it out in perfect detail, with a success which is most gratifying. I show you here the apparatus.

There is a leather sheath for the leg, almost meeting, laced together in front by a cord passing round the hooks. It may go above the knee or stop short of it. There is a similar sheath for the foot, and the two are connected by an isthmus of leather about an inch wide. From rings at the end of each sheath cords extend, in which india rubber is interposed. A strong common india rubber band answers perfectly well, for the elastic

The upper figure represents the shape of the brown paper pattern, and of the leather, cut in correspondence with it. The dimensions are approximate, varying with each leg. D is the narrowing for the knee, that the leather may not press on the bone at the sides. B is the excavation on each side for the ankle with the narrow connecting piece of leather, which is the special feature of the contrivance. This, 1 to 1½ inches wide, and no longer than is needful, affords, lengthwise, adequate support to make the pull from above act down on the foot piece on which the pull acts. At H and G are the rings for the cords. The piece of leather above the knee is not necessary, but is of use when there is a tendency to flexor contracture of this joint also, as described in the text.

force, as I said, does not need to be great, it needs only to be constant. The slightest constant traction is effective, but constancy, under varying mechanical conditions, can only be secured by elastic traction. It is remarkable how well the muscles bear such traction if only it is gentle. To the more sensitive skin, it soon becomes unendurable, but the impressions from the afferent muscle nerves only act on consciousness, so as to cause "sensation," when excessive, and even long-continued gentle extension seldom causes muscular discomfort.

The new feature which it embodies is this. The part of the apparatus for the leg *from* which the traction is made is continuous with that for the foot *on* which the traction is made. The two are

continuous by a narrow piece of the leather, which will bend so as to permit the traction to increase the flexion of the ankle, and so to overcome the shortening which has occurred, and which yet presents longitudinal resistance. This resistance is enough to make the source of traction, and, that on which it is exerted, effectively one. Instead of the pull being from the leg itself it is from the apparatus that the force acts on. Continuous traction from the leg itself—which, however slight, becomes unendurable by persistence — is thus avoided. The traction is exerted from a part of the same structure as that on which it is exerted. This piece of leather, narrow and short as it is, affords sufficient support, and no pressure is caused on the leg itself.

Another advantage of this apparatus is that it is easily made by any practitioner. A piece of leather must be cut out, of the shape which I now show you—two ovals connected by a narrow piece. It is best first to take a sheet of brown paper and cut out a pattern to fit the leg and foot of the patient. The leather must then be softened in water, warm at the last, so that the leather is warm when it is applied to the leg. It must be bound on the leg by cord or bandage, and then allowed slowly to dry and harden. The leg should be swathed to prevent chill from the drying. If thought desirable, oil silk may be placed next the skin. When dry and hard it is taken off. It has only to be lined with wash leather, and any shoemaker will insert in it the necessary hooks and rings. A piece of cord and an elastic band complete it. It may be made in rough but perfect efficiency, with holes and tapes or cords, and a penny elastic band for traction.

Mr. Hawksley, the well-known surgical instrument maker of Oxford Street, has made a copy of this apparatus, finished off with all due skill, and therefore more sightly than that which I show you, although it could not be more effective. He has been good enough to place it in his shop for any one to examine. As the only apparatus known to me that is really effective for preventing a grave evil, and even removing it when it has occurred, at least when it has not reached an extreme degree, and as one that is easy to make in perfectly serviceable form, I think it deserves to be widely known. It should be adopted in all cases in which a patient has to be kept in bed, and the weight of the foot leads to persistent extension of the ankle-joint, whatever be the cause of the condition, if

there is the slightest evidence of commencing contraction of the extensors, or if there is even reason to suspect that it is likely to come on.

But I have found that it produces another effect which I did not anticipate. When the leather is carried a little above the knee, as it is in the form shown in the figure, any tendency to flexion of the knee is counteracted, and with it flexion of the hip. When a disease that can be recovered from leads to such flexion, prevention is of great importance. When it occurs as the sequel to extensor spasm, treatment has little chance of doing good. The flexor spasm then says unequivocally, "Hope is at an end."

A CLINICAL LECTURE

ON

A CASE OF PYÆMIA.

Delivered at University College Hospital, Feb. 11, 1895.

By CHRISTOPHER HEATH, F.R.C.S.,

Holme Professor of Clinical Surgery.

GENTLEMEN,—To-day I have to speak to you about an interesting case of pyæmia, which unfortunately proved fatal. It is particularly interesting because you so seldom see cases of pyæmia nowadays. In my student days, I am sorry to say, pyæmia was the curse of the hospital, most of the large amputation cases dying of pyæmia a week or ten days after the operation. But now, thanks to antiseptics, and to the greater care which is taken of the patients generally, cases of pyæmia are extremely rare, and, if I had a case of pyæmia originating in my wards, I should be inclined to inquire of my house-surgeon as to why the case had gone wrong. I beg you to remark that this patient was admitted into the hospital as a case of pyæmia.

Before I go into the case I should like to tell you what pyæmia means, and I will read you what Burdon Sanderson says in his lectures on the "Infective Processes of Disease" (*Brit. Med. Jour.*, December 29th, 1877):—"What I mean by septicæmia is a constitutional disorder of limited duration, produced by the entrance into the blood-stream of a certain quantity of septic material. It must, therefore, be regarded not so much as a

disease, but as a complication, differing from pyæmia not only in the fact that it has no necessary connection with any local process, either primary or secondary, but also in the important particular that it has no development. Pyæmia is a malignant process which goes on and on to its fatal end ; but in the case of septicæmia, inasmuch as the poison which produces it has no tendency to multiply in the organism, there is no reason why the morbid process should not come to an end of itself, unless either the original dose is fatal, or a second infection takes place from the same or another source. The one is septic intoxication, due to the absorption of chemical products of decomposition, exceptionally attended by local mischief; the other embraces a group of diseases of which one, at least, is due to the multiplication in the system of a pathogenic organism, and in all of which metastatic inflammations so frequently occur that their presence is held to stamp the disorder as pyæmic, even when no evidence of a germ can be found."

The case which we have to consider to-day was one of pure pyæmia. A married woman, 24 years of age, was admitted to the hospital on December 28th, 1894. On November 18th the patient was confined of a full-term child after a difficult labour, with instruments. She went on well until about the end of November, when she noticed that her left knee was slightly swollen. The swelling increased, and the knee became very painful. Her doctor painted it with iodine and applied mercurial ointment, but as the knee continued to get larger he advised her to come to the hospital.

A week before the confinement the patient had been in bed with what was said to be influenza. The temperature went up shortly after the confinement, and on the sixth or seventh day it was decided to clear out the uterus, which was done on the ninth day by curetting it and syringing with iodine lotion. The discharge was not offensive, but a small piece of placenta was removed. The next day the temperature stood at 103° ; on December 1st it fell to 99.8° ; the next morning it rose to 103.8°, and it was on the 1st of December that the pain appeared in the knee. Between the 2nd and 18th of December the temperature varied, was normal on three occasions, and generally ranged between 100° and 102°. The first child was born two or three years ago, when there were no instruments used, the delivery being easy. The patient had a miscarriage at two or three

months in November, 1893, and had a second child in November, 1894, from which illness the present case dates.

When she was admitted to the hospital, we found a young woman well developed but rather pallid and of a yellowish tint ; she was in bed, and one knee was enormously swollen. There could be no question that the knee contained fluid, and from the history of the case I was quite certain that it must be pus. Accordingly, we gave the patient an anæsthetic, and made a free incision on each side of the affected knee, and let out twenty-seven ounces of pus. Large drainage-tubes were inserted, and pushed well up into the pouch of the joint. The joint was syringed out with a weak carbolic lotion, and dressed with tarred oakum pads. So far as the knee went, nothing could have done better. It was syringed out twice a day, and the tubes were kept in for some time, and then removed. Really, so far as we could see, everything went well with regard to the knee. The wounds practically healed, and when the patient died the continuity of the joint had been quite restored. But the patient never rallied as I hoped she would have done. The best proof is the temperature table. You will see that the temperature at the operation was 101°, then it dropped, then oscillated for the next day or two, after which it rose to 102°. It again worked up and down, and on the 3rd of January it reached 103.5°. Against that, in my own handwriting, is the note "Christmas-tree." This was because I thought and hoped that the excitement and bustle of the ward due to the Christmas-tree festivities might have sent her temperature up. But the temperature did not come down; as you see, it kept up to between 102° and 103°. It did once drop just below the normal, but on the whole it has maintained the characteristic rise and fall of a blood disease. And here, at the end, when the patient was much weakened, we find at times the temperature falling below normal. She at last died on February 4th, having been in the hospital thirty-nine days.

You have all seen the patient, and I hope you have studied her appearance, because it was very characteristic. Now, cases of this kind are almost hopeless from the first—that is to say, if they are *bona fide* cases of pyæmia. I have seen cases of pyæmia recover, but the majority are almost certain to die. But that does not absolve us from the necessity of trying to save them if possible,

and the way to save them is to evacuate the pus and to take the strictest precautions to keep the patient's wound aseptic, and then to adopt a stimulant and tonic plan of treatment. The first time I saw the patient, after opening the knee I ordered her to have 5-grain doses of quinine, which she was to take every four hours, and I put her on six ounces of brandy at once. In giving brandy you should not give it by tablespoonfuls, but by teaspoonfuls, since it is better for a patient suffering from blood-poisoning that the necessary stimulant should be taken in small but repeated doses. This patient had two teaspoonfuls of brandy every hour or two hours, according to circumstances. A teaspoonful of brandy every hour for twenty-four hours equals three ounces *per diem*, so that two every hour make six ounces. We had afterwards to increase the amount to nine ounces in the twenty-four hours, but we did not exceed that. When I was a student, the late Dr. Todd was promulgating the doctrine that stimulant treatment was the treatment for all acute diseases, and in some ways, no doubt, he carried his views to excess; but, at any rate, he taught surgeons that brandy or some form of stimulant is *the* medicine in cases of blood-poisoning. I am quite sure of this, that I have seen cases which have been saved by stimulant treatment, particularly cases of septicæmia, and occasionally a case of pyæmia. In the present case we did all that could be done, getting her to take all the nourishment she could, and giving her stout in addition to help the food down.

We were constantly on the look-out for secondary abscesses. I had her lungs carefully examined; I examined her abdomen, and found she had a large liver. But it was not till we were in the post-mortem room that we became aware that there was a secondary abscess in the region of the shoulder. Now that abscess, I am quite sure, must have formed within forty-eight hours of her death. If it had been formed before, I am certain it would have been noticed by the nurses or the sister. The patient was in a state of coma for sixty hours before death, and it is nearly certain that the abscess formed within the last two days of the patient's life. We opened the abscess in the post-mortem room, and found that it contained about six ounces of thick green pus, of peculiar odour. The abscess was lying beneath the deltoid, and also beneath the periosteum of the humerus, the bone around being necrosed. The

shoulder-joint was not diseased, as the abscess was outside it. As to the knee, we examined it, and found, on turning back the skin, that there was a small collection of matter on the inner side, below the joint which had been laid open. This abscess was in the tissues which had not been interfered with by the two incisions at the operation. When we came to open the knee-joint we found it as healthy as could be. My fingers had been in the joint, my house-surgeon had his fingers in the joint, and we had drainage tubes in it, yet, notwithstanding all that, there was not the slightest change in the synovial membrane or in the cartilages—in fact, there was really nothing the matter with the joint.

We went on to see if there was any trouble elsewhere. The liver weighed 6 lbs. 4 ozs., and was soft, large, and pale; it did not present the appearance of a hob-nail liver, and yet I cannot help thinking it was connected, in some sort of way, with drink. Taking into consideration the state of the liver and kidneys, I am of opinion that the woman must have indulged in alcohol. We then found that the spleen was enlarged, and slightly softer than normal. The pericardium contained very little fluid, and the heart was normal. The right lung was also normal, while the left showed a small area of congestion near the base at the back. The pleuræ were healthy.

Now we come to the important point as to what was found in the pelvis. The uterus measured $2\frac{3}{4} \times 1\frac{3}{4}$ inches, and, except for a few peritoneal web-like adhesions attached to the posterior surface, appeared to be quite healthy externally. On laying the uterus open, the placenta seemed to have been attached to the upper part of the posterior wall, and the placental site appeared to be healthy. On section, the uterine wall appeared to be normal for the period after delivery. There were no thrombi to be seen in the venous sinuses of the uterine wall. The Fallopian tubes were normal. The left ovary contained a *corpus luteum*, $\frac{1}{4}$ inch across. The ovaries were healthy. There was no lesion of the vaginal mucous membrane, but the cervix had been torn bi-laterally. I think that is most important. It seems to me that the mischief must have spread from that, rather than from the patch of retained placenta. I should imagine that little bits of placenta come away without much harm resulting sometimes, and it really appears as if the retained portion of the placenta was not the source of the evil, as the sinuses of the uterus were per-

fectly healthy. The left uterine veins were throm-bosed in the broad ligament, and contained broken-down clot. A similar clot was present in the internal iliac, the common iliac, external iliac, and femoral veins, as far as the middle of the thigh. Their contents were more purulent than those of the uterine veins. There was slight thick-ening of the tissues in the pelvic wall, round the left internal iliac vein.

Although, therefore, this patient survived her confinement from November 18th till February 4th, the mischief was entirely connected with the confinement, and she died from thrombosis of the veins and from pyæmia—the result of the breaking down of these clots. In this preparation from the museum you see veins from a leg with thrombosis and phlebitis. The patient died from erysipelas and pyæmia after amputation of the great toe. Remember that the mere formation of thrombi in a vein is by no means a very serious thing. We are constantly having patients with thrombosis of the saphena vein, an ordinary case of varicose veins in which thrombosis may occur accidentally. These get perfectly well, because the clot is not septic. A septic clot is sure to break down, and a condition of pyæmia is produced.

Of course the word "pyæmia" is a misnomer. It was formerly thought that pus cells could be seen in the blood. The early microscopists mistook the ordinary white corpuscles of the blood for pus cells, and it was said that pyæmia could be detected by seeing the cells floating in the blood. We know now that pus does not circulate in the blood; but that the tendency is for pus to be secreted, owing to the poisoning of the blood circulating through the body. Especially is this tendency most marked where there is great vascularity. You therefore expect this secondary deposit in a large joint, and the knee-joint is one of the largest. Next to the knee-joint it may be found most com-monly in the shoulder, and very commonly we find secondary abscesses in the liver; but I had this liver very carefully searched, and could find nothing of the sort. So also in the spleen, in the lungs, and in the kidneys these abscesses some-times occur, but there was no evidence of that.

I have been speaking till now of fatal cases of pyæmia; but a year ago we had in this hospital a case somewhat similar, which, however, was not fatal. The patient was a young woman of 22, married. She had never had any illness before, and had a child on December 27th, 1893. Four

days afterwards she noticed that her hand and arm were swollen and very painful. This gradually grew worse till the following week, when an open-ing was made in her wrist by her doctor—I think it was her left wrist—a large quantity of pus and blood was drawn off, and a fomentation applied. The swelling in the arm subsided, and the patient felt a good deal easier. On the 13th January, 1894, the patient began to feel some pain in the right hip, which prevented her walking; it was swollen and very tender. She had rigors soon after she had noticed the swelling of the arm, and had these attacks two or three days before the incisions were made in the wrist, and they lasted the entire day. She was admitted here on January 27th, 1894. This happened to be a Wednesday, and I did not see her until the following Friday, the 29th. When I saw her, the condition was that of a fairly well nourished woman of 22, with no excess of subcutaneous fat, the face was rather pale, mucous membrane also pale. On admission her temperature was 103°, which fell early on the 28th January to 100°. It rose in the evening, however, to 102.4°, and on the morning of the 29th of January was 100.4°. There was no tenderness on deep pressure above the pubes, the uterus could not be felt through the abdomen, and on vaginal examination nothing could be felt abnormal; the breasts were typical of lactation, and not pain-ful. The left wrist was swollen and tender, and there were two sore places covered with rather pale granulations, one at the base of the first metacarpal, and another an inch in front of it. The anterior sore was an inch and a half across; the posterior sore seemed to lead into a sinus, from which turbid fluid escaped on moving the hand. A probe passed the distance of one inch inwards, and touched on bare carious bone. The right hip was very painful and tender, and was most com-fortable when extended and inverted. It could be flexed, but movement of the joint was painful, though not severely so, and the joint was by no means fixed. Jarring the head of the femur against the acetabulum did not cause pain, and there was no grating in the joint. The front of the joint was slightly tender, but was not swollen. The gap between the ischial tuberosity and the great trochanter was filled up by an extremely tender swelling, in which fluctuation could be obtained.

Here was a case with a history of a confinement, with an abscess in the left wrist, and an abscess in

the right hip, and when I saw the patient her condition was very serious. Her temperature was then up to 103°, and altogether she was exceedingly ill. It became a nice question—Was that matter in connection with her hip-joint, or not? I concluded that it was not in direct communication with the joint, but was outside it. In arriving at this conclusion I, in the first place, went by the movements of the hip, and in the second place by the inversion of the thigh. If the joint was full of matter, I should have expected to find it everted; but, on the contrary, it was inverted, and there was also the soft swelling between the ischial tuberosity and the great trochanter. So I had the patient brought down into the theatre, an anæsthetic was given, and I punctured with a large aspirator needle and found that I got matter. Thereupon I made a free incision and introduced my finger, and although the matter was under the glutei muscles the joint itself was not involved, and I carefully abstained from opening into it. Then I turned my attention to the wrist. I found the scaphoid bone was loose and carious, and I therefore took it out, but the other carpal bones were left. I had the hand put up on a back splint with the fingers extended, and I hoped the result would be a useful hand. As regards the buttock, after gently scraping it with a sharp spoon, a large drainage tube was put in after thoroughly syringing out with a carbolic lotion. We then pursued the same treatment internally as in the other case—five grains of quinine, with thirty minims of dilute hydrobromic acid, in an ounce of water. This was at first ordered three times a day, and then I find I altered it myself to every eight hours. I did that because when you give quinine in large doses you want the doses distributed over the whole twenty-four hours, and that is an important point. I also gave her four ounces of brandy *per diem*. That woman recovered; she got perfectly well, and went out. The abscess at the hip closed and filled up, and the abscess at the wrist closed also. Although she had some little deformity and slight stiffness, she went out perfectly able to walk and in very good health, after being in the hospital fifty-two days. So you see it is possible for a patient to recover, even after pyæmia, though of course a good deal depends on the previous condition of the individual.

Let me give here, in illustration, another little extract from the article on pyæmia:—"It is even probable, as showing the influence of these pre-disposing causes, that the tissues of the human body, so long as they are healthy, possess the power of resisting the action of these germs—perhaps even of destroying them. Burdon Sanderson has shown, with regard to bacteria producing the poison of septic intoxication, that even if they do enter they have no effect on the healthy organism; and this principle may have a much wider range of application, serving, for example, to explain the susceptibility to scarlatina of patients suffering from traumatic fever." Let me here say that neither of these women was in perfect health—they had both gone through the perils of child-birth, and were therefore predisposed to the attack of an unhealthy action. We all know, or ought to know, that puerperal women are especially liable to an attack of any disease which is near them, and it is for that reason we exercise such great care. No man is justified in going to a puerperal woman's bedside after having seen recently any infectious case; the disease would be sure to be taken, and the patient would be sure to do badly.

Let me say one word with regard to these cases, whether they be septicæmia or pyæmia, you cannot go wrong if you use vigorously supporting and stimulating treatment. The younger generation of medical men does not appear to be always clear on this point. Some years ago I was called to see a gentleman who had clearly got pyæmia or septicæmia, and I was inclined to believe it septicæmia. When I met the doctor I asked what the case was. He said, "A bad case of septicæmia, and I am afraid he will die." I saw the patient, and I also was afraid he would die. I asked the doctor what he had been doing for him, to which he replied: "I am giving him one grain of carbolic acid three times a day." I did not say anything, it would have been of no use; the medical man evidently had not grasped the situation. I only said it was very good treatment so far as it went, and added, "If you will get home as soon as you can and send a messenger over with some five grain powders of quinine, I will, in the meantime, get up some champagne from the cellar and see what we can do." Now, here was a rich man, dangerously ill, with any amount of stimulant ready to hand, and yet he had never been offered any. Well, I got up the champagne and gave some to my patient, at the same time impressing on his wife to go on with the champagne, and to add brandy to it, giving him soup and everything

nourishing. But the end of it was the poor fellow died. I did what I could to cover the retreat, but it was not of much use, for the friends of the patient, naturally enough, said, "It is all very well, you may say what you like; the doctor may have done all that he could according to his knowledge and power, yet it is perfectly obvious that Mr. Heath took a different view, as anyone can see from his change in the treatment." What is it possible for anyone to say in a case like that? Fortunately the thing blew over, but the fact remains that that patient's life was probably lost from the case not having been taken in time.

In the case of the woman with the bad knee, the gentleman who attended her had obviously not recognized what was the matter with the knee. You can see this from his having used iodine and Scott's dressing. I do not like the use of iodine myself, and, as for Scott's dressing, it is a useful remedy only in its proper place; but where you have an acute case, with fluid in the joint, the probability is that there is pus, and there can be no question about the treatment. You will have noticed that in the second case I used an aspirator needle, though I was sure there was matter there. I should strongly advise you, before you lay open a joint, to use an aspirator needle, to make quite sure it is matter before making a free incision. In aspirating use a large needle. I was once myself unintentionally misled by a practitioner over a case of perityphlitis. He assured me that he had aspirated and found pus, but when I made an incision there was none. The mistake arose from the practitioner using a hypodermic needle. This had been carefully cleansed with carbolic lotion, and serous fluid passing through a small needle recently cleansed with carbolic gives an appearance very like pus, and probably led to the mistake. The proper treatment is to open freely every abscess in a case of pyæmia as soon as it is formed. I regret we did not know of the abscess in this poor woman's shoulder, but there is no doubt it formed very late in her life, and the omission made no difference. The only wonder is that she lived as long as she did. More could not have been done for her, as the case was hopeless from the beginning.

It is worth while to always make a good fight, however, because every now and then you will succeed in pulling a patient through, although I am sorry to say our efforts in this case were of no avail.

A CLINICAL LECTURE

ON

TWO CASES OF MALIGNANT DISEASE OF THE LIVER.

Delivered at Guy's Hospital, Feb. 2, 1895,

By W. HALE-WHITE, M.D.,

Physician to the Hospital.

GENTLEMEN,—The first patient whom you see is 41. He has never been abroad; he is said to have had syphilis, but this is doubtful. For the last fifteen years he has been a barman. In May, 1894, his urine became dark, his skin yellow; he had xanthopsy, and his motions were clay-coloured. He came into the hospital under Dr. Taylor; his liver was then uniformly enlarged, and extended within two inches of the umbilicus; it was not tender. The gall bladder could be felt. There was jaundice, bile in the urine, and the motions were clay-coloured. He finally left the hospital on October 26th, his condition being unchanged.

January 1st, 1895. He was very weak; he noticed that his feet and abdomen began to swell, so he came in on January 21st. The jaundice has varied in depth, but has been constant, and the motions have generally been clay-coloured.

On admission. He appears wasted; he is deeply jaundiced, of a dark greenish-yellow tint. There are many scratch marks, showing the itching due to the jaundice. He has some internal piles. There is no evidence of syphilis. The abdomen is uniformly distended, and gives the ordinary signs of ascitic fluid. On measurement we find that the vertical extent of the hepatic dulness has increased half an inch since he was in the hospital before. The edge of the liver can easily be felt a little above the umbilicus; it is uniform, and somewhat hard. The notch can be quite readily distinguished, and in the position of the gall bladder and attached to the liver is a soft globular swelling, the size of a Tangerine orange, apparently containing fluid, which we take to be the gall bladder. There are two very tender points on the surface of the liver—one over the left lobe, the other below the ribs in the mid-axillary line. The rectum is normal. The motions vary, but are often clay-coloured, and the bowels are open several times a day. There are no venous stigmata on the cheeks. There has been no vomiting or hæmatemesis.

There is no evidence that the spleen is enlarged. The urine contains bile and urates. Excepting that the apex of the heart is a little displaced to the left, there is nothing noteworthy about the chest.

The second patient, a man who died yesterday, aged 43, was admitted January 26th, 1895, for jaundice, wasting, and abdominal pain. His father died of a tumour of the testicle. He has never been out of England, and there is no evidence that he has ever had syphilis. On the whole, he has had excellent health until the present illness, but he has been liable to slight attacks of indigestion, lasting a day or so and accompanied by some pain, but no sickness; and clearly from his account they have not seemed to him to be of much importance. Six weeks ago he began to get weak, and for three weeks this weakness increased; he then found that his appetite failed, and that his urine was dark. He now had to give up work, and he began to feel a cutting pain in the hepatic region increased by coughing. A fortnight ago he was so ill that he went into King's College Hospital, but he was only there nine days. Two or three days ago he began to get yellow, and his urine became very dark.

On admission. He is much wasted, considerably jaundiced, and looks very ill. His weight is 9 st. 4 lbs.; in health it was 12 st. The epigastric angle is wider than normal. The liver is obviously very much enlarged. Its lower edge is hard and reaches very nearly to the umbilicus, just above which is the hepatic notch. The upper limit of hepatic dulness is normal in the nipple line, but does not fall as it should, for it reaches the upper border of the sixth rib in the mid-axillary line, from which it falls to its normal position at the base of the right chest. On the surface of the liver two rounded prominences can be felt—one about two inches above the umbilicus, the other just below the eighth and ninth costal cartilages. A rub can be felt and heard over the surface of the liver when the patient breathes. The spleen cannot be detected. There are a few râles at the right base. The apex of the heart is displaced up a little. He is occasionally sick; the vomit contains no blood, but hydrochloric acid is present. The rectum appears normal; the motions are clay-coloured. The urine contains bile and a very large amount of urates. On January 28th he showed signs of bile poisoning or cholemia, the chief symptoms of which were unconsciousness and drowsiness, with at times slight delirium. The liver increased in size, so that before he died on February 1st, it was below the umbilicus. I particularly want you to notice the increase of normal dulness upwards in the mid-axillary line, for being dome-shaped we concluded it was not due to fluid in the chest, as in that case its upper limit would have been a horizontal line; therefore it must have been due to something in the liver or between it and the diaphragm, and as the liver was obviously diseased we concluded that the former supposition was correct.

It is obvious that there are many points of view from which we might consider these two cases, but in the short time of an hour we can only take one. I propose that we should in each start with the undoubted fact that the liver is enlarged. Whenever you are examining the liver you should always make a habit of remembering that it may appear enlarged when it is not; for instance, it may be pushed down by fluid in the chest or by tight lacing, and in children it is in health proportionately larger than in adults; or, again, other abdominal tumours, such as a thickened omentum, may, by their proximity to the liver, give an impression that it is enlarged. It also may appear much smaller than it really is, for the enlargement may be hidden by an accumulation of gas or fluid in the abdomen, or by emphysema in the chest; but, after bearing all these fallacies in mind, we came to the conclusion that the organ was, in both our patients, undoubtedly enlarged, the chief reasons being that the enlargement and the dulness were such as we should naturally expect with a large liver; the lower edge felt like, and occupied the position of, the edge of an enlarged liver; it moved freely with respiration; the patients had other signs of hepatic disease, but none of any of the fallacies we have mentioned. What, then, are the causes of enlargement of the liver? They are:—

1. *Passive Venous Congestion.* Generally from heart disease or bronchitis. I have often demonstrated this to you and have shown you that the liver is uniformly enlarged and frequently very tender and painful, the pain being much relieved by leeches and a dose of calomel. However, in these cases there was no cause for venous congestion, and the jaundice was too deep.

2. *Passive Portal Congestion.* This gives rise to only slight enlargement of the liver, but it may occur if a habitual discharge of blood from piles

is cured by operation, or it may be seen in those who suffer from chronic constipation.

3. *Active Congestion.* This is seen chiefly in India where the combination of high temperature with irritating articles of food gives rise to enlargement and tenderness of the liver. Do let me beg of you never to say, for a diagnosis, that a patient has congestion of his liver. It is a popular phrase, and even if you believe it to be present—which it often is not when the patient declares it is—you sail very near to the class of ignorant quacks if you do not try to find out the cause of it, and from that you should make your diagnosis.

4. *Malaria.* 5. *Yellow Fever.* These causes of enlargement, obviously, do not apply to our present patients.

6. *Leuchæmia.* 7. *Hodgkin's Disease.* 8. *Pernicious Anæmia.* 9. *Diabetes.* There was no evidence of any of these, in all of which the enlargement of the liver, if it occurs, is uniform, painless, and unaccompanied by jaundice.

10. *Fatty Liver.* In our cases the enlargement was very extreme for a simple fatty liver. Moreover, a fatty liver feels soft, is painless, smooth, uniform, does not evert the ribs, and does not cause jaundice or ascites.

11. *Hydatid.* In this disease of the liver there are localised tumours of it, and, therefore, the localised enlargement upwards and the lumps on the anterior surface in the second case, might have been due to hydatids, but there was no thrill, the patient was far too ill, the course of the disease was too rapid. Also jaundice and clay-coloured stools are usually absent in hydatid, for it is rare to get a hydatid tumour pressing on the bile duct, and, therefore, we decided against hydatid. In the first case the enlargement was too uniform, and, again, the jaundice and clay-coloured stools showing pressure on the bile duct, helped us to exclude hydatid.

12. *Tropical Abscess.* 13. *The Single Large Abscess found in those who have never been abroad.* 14. *Suppurating Hydatid.* 15. *Actinomycosis.* 16. *Tubercular Abscess.* The long duration of the first case and in both the absence of pyrexia, rigors, and other general and local signs of suppuration enabled us to exclude these conditions. You all remember that last year, in a clinical lecture, I pointed out that sometimes the solitary abscess which occur in those who have never been abroad is associated with ulcerative colitis. Actinomycosis is very rare. You will find a case

recorded by Dr. Taylor in the Guy's Hospital reports. Tubercular abscess is even rarer, and most supposed cases of it are really examples of actinomycosis; still, an undoubted example has been recorded by Mr. Mayo Robson. Remember that pyæmic abscesses of the liver are never large enough to give rise to tumours that can be detected during life.

17. *Perihepatitis.* This only causes an enlargement of the liver sufficient to be detected during life when the hepatic capsule is very much thickened. It then forms an opaque covering for the liver, as you can see in this specimen, and the lower edge of the liver gets turned up on itself, consequently the edge you feel is thick and rounded. But the liver in neither of these cases has such an edge, and in both it is far too big for the increase in size to be due solely to thickening of the capsule.

18. *Obstruction of the Common or of the Hepatic Duct.* When this occurs the bile gets dammed back in the liver, and so may cause the liver to be a little enlarged, but in both our patients the enlargement is far too great for this to be the sole cause.

19. *Lardaceous Disease.* Uncomplicated lardaceous disease of the liver causes the organ to be uniformly large—often very large—with a hard edge. There is no pain, tenderness, nor jaundice, and if the enlargement is very great there should also be enlargement of the spleen and albuminuria, from lardaceous disease of the spleen and kidneys. Also a history of suppuration or syphilis is usually obtainable. You will at once see that our cases cannot be examples of a simple lardaceous liver.

20. *Hypertrophic Cirrhosis.* In this disease the enlargement is uniform, the liver feels hard, and sometimes smooth, but often distinctly uneven; it is rarely tender. We did not think our second patient had cirrhosis because the enlargement was not uniform. He had considerable pain, the liver enlarged rapidly under observation, the jaundice was deep, and there was no history of alcohol.

In the first case the question is far from easy; the fact that the patient has been a barman is a very strong point in favour of the view that he has cirrhosis, and also I have repeatedly urged to you at the bedside of other cases that in cirrhosis ascites appears very rapidly and often quite unexpectedly. Time will help us much in our present patient. He has now had the ascites for a month. Out of twenty-two cases of cirrhosis with

ascites, five were dead within a month from the first day on which the patient noticed the abdomen to be swelling, and the average duration of life was only two months. The œdema of the feet is also a point in favour of cirrhosis, and I have often given you reasons for believing that the ascites and swelling of the feet are not mere pressure effects. A patient with marked cirrhosis of his liver may present no symptoms at all of it, for it is often found accidentally in the post-mortem room; but all patients who have a cirrhosed liver are liable to the more or less sudden supervention of symptoms which rapidly increase in severity, and usually soon end in death. The chief are ascites, swelling of the feet, and a general feeling of illness and drowsiness.

Now while it is true that as our first case conforms to much of this description, if we diagnose cirrhosis we shall have an explanation of many of his symptoms. Nevertheless, the following points must be urged against such a diagnosis, namely, he has clay-coloured stools, his jaundice is deep and is getting greenish, and his gall bladder is dilated—all of which mean that there is some actual obstruction to the common duct which never occurs in uncomplicated cirrhosis. Further, death usually takes place in cirrhosis soon after the supervention of jaundice, but this patient has had it for months. Also it is specially those cases in which the liver is big that death takes place soon after the patient complains, and before ascites developes. Then, again, the spleen is enlarged in half the cases of cirrhosis, but it has always appeared normal in this man, and it is just in those cases in which the liver is big that it is most often enlarged. Lastly, the diagnosis of cirrhosis does not explain the tender spots on the liver. There are no other signs of cirrhosis, e.g., gastric, the look of the face, etc. I should, however, add that it is with the large cirrhotic liver that jaundice is most often seen; but it bears on this case to state that I found among 68 cases of hypertrophic cirrhosis of the liver, collected by Dr. J. A. P. Price in the Guy's Hospital Reports, jaundice and ascites only occurred 19 times, and in 29 neither symptom was present.

21. *Congenital Syphilis.* We may quickly dismiss congenital syphilis of the liver. It exists in two forms; the commonest is only met with in quite young children. The organ is slightly enlarged, hard, and of a grey colour from excessive growth of new interstitial tissue, especially round

the portal vein, which does not go on to the formation of contracting fibrous tissue, and therefore the liver is not like a cirrhotic liver. The second form is especially interesting to us, for we had a boy in Stephen who showed it admirably. The organ is enlarged, hard from the formation of new fibrous tissue, and irregular from the presence of many gummata. The boy was 6 years old. His liver reached nearly to the umbilicus; but I never read or heard of this condition existing after the age of 16.

22. *Acquired Syphilis.* When the liver is affected, gummata, ranging from a few up to fifty, and differing in size from a pea to a Tangerine orange, are deposited in it, and broad fibrous bands, it may be ¼ inch wide, appear running through the liver in various directions. As the gummata are absorbed or the bands contract we get a number of depressions on the surface of the liver, with fibrous bands radiating from each depression. These scar-like depressions are absolutely diagnostic of syphilis. Nearly always a syphilitic liver is more or less lardaceous, so that you can easily understand that the liver is hard, irregular, and enlarged. But we never for a moment thought our second case had syphilitic disease of his liver, for he was dying, the course of his disease had been very rapid, and he was jaundiced. In the first patient there may be some doubt, but on the whole I do not think his is a syphilitic liver, because, for its size it is not irregular enough, he is jaundiced, has ascites, and passes clay-coloured stools, which would show that a gumma or a lardaceous gland is pressing on his bile duct and portal vein; but both these are so excessively rare as to be only pathological curiosities. I show you a specimen of a gumma pressing on the portal vein. Then again, although you may have pain in a syphilitic liver from coincident perihepatitis, you rarely get tenderness, and lastly the man has no other signs of syphilis. Still he is having iodide of potassium on the chance that we are wrong in regarding the liver as non-syphilitic. Up to the present time he has not improved under it.

23. *Malignant Disease.* In our second case the wasting, the fact that the man was dying rapidly, that his jaundice was quickly becoming very dark, that the urine contained much bile, that the motions were clay-coloured, that the liver was very large, irregular, and with palpable masses on it, that it increased rapidly in size, all pointed so certainly to malignant disease of the liver that we

diagnosed it, feeling sure that we were right. Further, we were able to say for certain that he had secondary enlargement of the glands in the portal fissure, and that they were pressing on the hepatic or common duct, for deep greenish jaundice, much bile in the urine, and clay stools always mean obstruction to the flow of bile after it has left the liver.

Whenever you have made out that your patient has malignant disease of the liver you must always begin to search for the primary seat in some other organ, for the liver is so rarely affected with primary malignant disease that this diagnosis can never be made with any probability of success.

The commonest seat of the primary disease is the stomach, but in our patient there was no evidence that this organ was affected, and the presence of free hydrochloric acid in the vomit was against this view. The next most likely primary seat is the gall bladder, the growth being set up by the irritation of a gall stone, but we could get no history of this. The rectum was healthy, and no primary seat could be found anywhere. When you have no clue as to the primary seat it is very probable that it is in the head of the pancreas, and so we suggested that our patient had primary disease of the head of the pancreas, with secondary growths in the liver. If you look at these organs removed from the patient yesterday, you will see that this diagnosis was correct. Here is a large mass of primary carcinoma of the head of the pancreas; here are several enlarged glands in the transverse fissure of the liver which press on the hepatic duct, and so have caused the jaundice. The fluid found in the gall bladder was colourless, because no bile could reach that structure. The liver, you see, is enormously enlarged; it weighs 19 pounds, and it is crowded with masses of secondary growth. You see two especially large lumps on its anterior surface, which we felt during life. You see a large mass on the upper surface of the right lobe, which caused the dulness to be higher in the axilla than normal. You notice, too, that at some spots where a growth on the surface of the liver was in contact with the abdominal wall nodules of carcinoma developed in the peritoneum from contact, and you can easily see how the growths, rubbing together during respiration, produced a rub. Some of the growths are umbilicated, which is a common occurrence in hepatic cancer, and the depression may even be detected during life. Lastly, you notice a quantity of

hæmorrhage in the hepatic substance which partly explains the rapid increase in size of the liver which was observed during the few days we saw the patient alive.

With regard to primary carcinoma or sarcoma of the liver, it occurs once in about every thirty cases of malignant disease of that organ, but as in the twenty-nine cases of secondary hepatic growth, it will be possible to detect the primary seat during life in a certain number, when we cannot detect the primary seat the chances are somewhere about fifteen to one against the disease being primary in the liver. I have been very fortunate in seeing this form of disease, for I have met with three instances, and from these and others I have collected it appears that the main characteristic of it is its extreme rapidity, for if the patient survive four months it is almost certain that the disease is not primary in the liver, and he may die before there is time for jaundice to be present. The extreme rapidity in our second case, the recent appearance of the jaundice, and the rapid enlargement of the liver made us wonder whether the disease could be primary in the liver, but the rarity of this made us hesitate to make such a diagnosis.

The first case is much more obscure. Although as already mentioned there are some points in favour of the view that he has cirrhosis, yet we have been able to mention several symptoms that this diagnosis fails to explain. The chief are the clay-coloured stools and the dilated gall bladder, both of which would be easily explained if we believe that there are some enlarged cancerous glands in the transverse fissure of the liver pressing on the hepatic duct. Then, too, the fact that the jaundice is deep, of a green tint, and that it has lasted many months, are all much in favour of growth rather than cirrhosis, in which disease jaundice is usually slight, and does not last long before the patient dies. The presence of two tender spots is difficult to explain on the view that the patient has cirrhosis; but if we believe he has growth the difficulty disappears. Then, too, for some unexplained reason portal obstruction due to growth rarely enlarges the spleen, but that due to cirrhosis frequently does. This man's spleen is not, that we know of, enlarged. Either view would account for the ascites and for the lithates in the urine, so on the whole I incline to the belief that he has malignant disease of his liver, with secondarily enlarged glands in the transverse

fissure, and that as the symptoms have lasted some time there is a primary growth somewhere out of observation, possibly in the pancreas. It has been suggested that perhaps in this case malignant disease has occurred in a cirrhotic liver, but this is excessively rare, and the case is lasting a long time for such a combination. It is particularly important to remember that the rapidity of our second case is quite exceptional. Malignant disease of the liver is usually a slow malady, patients often living many months after the supervention of jaundice.

PSEUDO-APPENDICITIS.

An Abstract of a Lecture delivered in October, 1894,

By C. H. GOLDING-BIRD,

Surgeon to Guy's Hospital.

GENTLEMEN,—The fact that the extensive and strong adhesions that must result from chronic suppurative appendicitis should not more often than they do lead to subsequent and perhaps lifelong trouble, is somewhat astonishing; yet these sequelæ of the disease do at times occur, and may, when seen for the first time, be taken for actual attacks of appendicitis; and this is the more likely to happen when the attacks of pain and colic are accompanied by a tumour. Such a case I lately brought before one of our societies ;* and as contrasting with it, and yet in many ways resembling it, I think the following case should be put on record. The same diagnosis as in the case above referred to was given, namely, appendicitis, and the same operative measures were undertaken for its relief, but with the result of demonstrating that, despite the clearest of clinical evidence, the symptoms were due to a condition that had no reference whatever to the state of the appendix; and when the history has been perused, I think it will be conceded that no other diagnosis than that given could have been fairly entertained, and as a consequence, the treatment proposed was the best adapted to meet the requirements of the case.

John H., 33, a blacksmith, with a good family history, and none whatever of tubercle, came under my care in Guy's Hospital, in September, 1894. Though following a laborious trade he did not strike one as being a robust or particularly strong-looking individual.

* Clin. Soc. Repts., 1894.

In February, and again in May of 1894, he first had attacks of colic, pain, and vomiting, similar to that which occurred later on in August, and which then brought him under the notice of Dr. Vernon, of Ashford, who very kindly furnished me with a report of the man's condition.

The two earlier attacks, each of which lasted about ten days, passed off, leaving the patient apparently quite well, and not influencing, either one way or the other, the subsequent daily action of the bowels.

The attack in August Dr. Vernon thus reports upon :—" Pain in right inguinal region on August 22, 1894. T. 101. P. 100. Local tenderness in r. iliac fossa; rigidity of the right rectus abdominis muscle; no vomiting, no diarrhœa, but severe colicky pain in the cæcal region." (Vomiting had been a feature the two first attacks.) " Five days after first being seen I could make out," writes Dr. Vernon, " a small swelling, about the size of a Tangerine orange, in the line from the antero-superior iliac spine to the umbilicus; there was great local tenderness. For a week the symptoms continued with a daily M. and E. temperature of about 101° F., and the bowels were moved on alternate days with a saline purge. The temperature now sank to normal, and though the patient improved under treatment, neither the lump nor the local pain disappeared."

When he came under my care, one month after the last attack, the patient was to all appearances very ill, and complaining of much pain in the cæcal region; and an examination revealed all that had been seen before, except that the lump in the abdomen was now clearly adherent to the parietes, and in a somewhat lower situation than when first observed; over it, also, the skin was slightly erythematous.

The case seemed to be an undoubted one of appendicitis, with adhesions to the parietes and a threatening abscess; so on operating, with the full expectation of meeting with this condition, it was somewhat surprising to observe that the gut everywhere, including the appendix, was quite normal, nor was there any appearance of its ever having been inflamed; the serous covering was natural. What really presented itself was the omentum matted together at its lowest part, thickened and adherent at its extremity to the parietes in the situation of the tumour that had been felt and which it had caused; elsewhere the omentum was free and movable. The extra-

peritoneal tissues of the parietes at the point of adherence were infiltrated with a curdy pus. The omentum was now divided in its continuity between two ligatures, and the adherent part, together with all the thickness of the pus-infiltrated parietes over the area of adhesion, except the skin. After due irrigation the wound was united without drainage.

An examination of the parts removed showed no cause for the adhesion and suppuration; but as the pus was curdy in character, it is likely that a deposit of tubercle in the omentum was the origin. The point of adhesion did not correspond to the internal ring.

It should have been mentioned earlier that a rectal examination revealed some pre-rectal thickening, just as one may feel after pelvic peritonitis; but nothing was seen at the time of operation to throw light on this condition. The patient made an uninterrupted and perfect recovery.

In this case the cause of all the symptoms was the adherent omentum; and it seems to have acted, as will an irreducible omental hernia sometimes, by dragging upon the large bowel, interfering with peristalsis, and giving rise to severe colicky pains; and when to these symptoms we add the presence of tumour in the cæcal area and the absence of any hernial protrusion, the history of two other attacks, and a week's history of high temperature, the picture of appendicitis seems pretty complete. The presence of the swelling is a most reliable symptom, and Talamon,* writing on this subject, states it as his opinion that in acute cases there can be no mistake if there is tumour also; whilst in the sub-acute—to which this case was regarded as belonging—he only admits the tumour may mislead if it is not in the usual area in which the appendix is found. Mere appendicular colic (or a colic resembling it) which runs its course, like biliary colic, with great rapidity and severity,† but which yields perhaps to a simple dose of opium, might have been the only symptom in this case, and the man might never have come to operation but for the fact that the point of adhesion of the omentum had begun to suppurate; and thus by prolonging his illness, by giving rise to a week's high temperature, and by making the tumour both more evident and painful, both compelled him to seek relief and helped to confirm

* Talamon, "Appendicite et Pérityphlite," p. 187.
† Compare *ibid*, p. 111; and Jessop, "British Medical Journal," March 24, 1894, p. 627.

the diagnosis of appendicitis which was given. This case affords us a true clinical picture of appendicitis; and we must recognize the fact that clinically, at least, we may have appendicitis without an appendix — Hamlet with the Prince of Denmark left out. But fortunately for this and similar cases the treatment is the same, and surgical interference at the proper time alone offers the prospect of permanent recovery.

THERAPEUTICAL NOTES AND FORMULÆ.

Night-Sweats—Treatment.—The July number of the *Brooklyn Medical Journal* contains a valuable report on the treatment of night-sweats of pulmonary tuberculosis. Only individual drugs were employed for the purpose named, and while many were subjected to the test during a period of five years of clinical experimentation, but eight proved successful to any practical degree. Among those having more or less therapeutical value were aromatic sulphuric acid, camphoric acid, chloralamid, muscarine, oxide of zinc, atropine, tincture of belladonna, and agaricin. The latter drug, according to the reporter, Dr. Henry Conklin, ranked first, and concerning it he comments as follows:—

This was the most successful of all the drugs. It produced most excellent results in young subjects. Under its use the skin remained in a dry condition, without suspicion of any kind of cutaneous activity. It was very successful in cases where, during its use, the sweating had disappeared and had returned after the drug had been discontinued for a time. Repetition did not weaken its power. Of all the remedies, it acted best in the first few administrations. Subsequent ones sometimes failed. It can be used for any length of time, and has no disadvantages. Agaricin was given in pill form, gr. 1–12; one pill at bed-time, or a pill late in the afternoon, and a second in four or five hours. This remedy diminished the sweating in one-eighth of the administrations, stopped it in three-fourths, and failed in the remainder.

Furunculosis.—Beer yeast, two to three tablespoonfuls daily before meals, in glass of beer. In recurrent cases, treatment should be prolonged for fifteen days. Only inconvenience, diarrhœa, which is rare.—(*Repertoire de Pharmacie.*)

Whooping Cough and Antispasmine.—Truhwald has employed this new remedy in 200 cases of pertussis in Vienna. The remedy is a fine powder, consisting essentially of salicylate of sodium and a compound of sodium with narceine, very soluble in water, forming a slightly bitter colourless solution. It is a powerful antispasmodic, useful in all forms of paroxysmal cough. The prescription recommended is a 5 or 10 per cent. solution in cherry-laurel water, of which 3 or 4 minims may be given three times a day to an infant under six months, 5 to 10 minims to a baby between six months and a year, and so on in proportion to age. The drug decomposes on exposure to light and air, and so must be kept in the dark in a well-stoppered coloured bottle.

(*Le Prog. Med.*)

Treatment of Granular Conjunctivitis.—The *Journal de Pharm. et Clinic* has the following :—

℞ Mercuric oxide   ...   ... gr.iij
   Zinci   ...   ... ⎫
   Thymol   ...   ... ⎬ āā gr.iss
   Muriate of cocaine ... ⎭
   Camphor ...   . gr.ss
   Vaseline ...   . ℨj
  M. Ft. unguentum.

Antipyrin Mandelate.—A new preparation of antipyrin, formed by adding to the latter mandelic acid. Tried in some sixty cases of whooping-cough, with failure in only two cases. Dose for infants under 1 year, ⅞ to 1¾ grains ; for children between 3 and 5, 4 to 7¾ grains.

(*Münchener Med. Woch.*)

Gout.—Local application: bicarbonate of sodium, 1 part ; linseed oil, 9 parts. Wrap affected joint in a piece of liniment soaked in this mixture.

(*College and Clinical Record.*)

Synovial Cysts.—Wash and disinfect the skin. Inject with a Pravaz syringe, previously sterilized, several drops of pure tincture of iodine into the most prominent portion of the cyst, taking the precaution to draw the skin lightly to one side, so that the puncture in it will not correspond to that in the wall of the cyst when the needle is withdrawn. Insert the needle deeply enough to penetrate the centre of the cyst, and evacuate by a single movement the number of drops intended, so that it will penetrate into the interior of the cyst and become mixed with its contents. It is sometimes necessary to exercise considerable pressure, particularly when the cyst is hard or thick. Leave the needle in place for a few seconds after the injection, and then draw it out with a single rapid movement. Compress the surface with the index finger to prevent the escape of the iodine. From 5 to 12 drops of the remedy are sufficient, according to the size of the tumour. Apply over the site of puncture a tampon of aseptic cotton, cover the joint with several layers of cotton, and secure it with a bandage sufficiently tight to compress the tumour and immobilize the joint. Remove the bandage on the third or fourth day, when the cyst will be seen to have greatly diminished in size. If necessary, continue the depression and immobilization for a few days longer. If the cyst has not diminished in size, owing to its calibre or volume, make a second injection of iodine, using a few drops more than at first. This will ensure a definite cure within from six to twelve days.

(*Archives Générales de Médecine*).

Treatment of Erysipelas.—Arnozan strongly recommends (*Arch. de Méd. et de Pharm. Milit.*) the following treatment :—Quinine is administered in doses of 8 to 16 grains, in accordance with the temperature, 4-grain pills being given three or four times daily, so that the patient is kept constantly under the influence of the drug. Over the affected surface is applied an ointment made up of—

℞ Bichloride of mercury ... gr.j
   Lanolin
   Vaseline, of each   ... ℨss

Of twenty-five cases thus treated there was frequently noted a prompt arrest of the eruption, or, in case this failed, marked alleviation of the symptoms. The temperature fell in three or four days, and the average duration of the attack was five to seven days.

Dyspnœa of Phthisis.—Dr. Bernheim recommends the following :—

℞ Caffeine citrat. ...   ... gr.iij
   Sulphuric æther ...   ... ℨv
  Injected hypodermically ℳxxx morning and evening.

LECTURES

ON

SOME OF THE LESS STUDIED FORMS OF DYSPEPSIA.

BY

W. SOLTAU FENWICK, M.D. Lond., M.R.C.P.,

Assistant-Physician to the Evelina Hospital for
Sick Children, etc.

I. HYPERACIDITY.

GENTLEMEN, — Modern researches upon the pathology of digestion have brought to light many interesting and important facts. It was formerly the custom to regard most of the symptoms of dyspepsia as the outcome of a deficient secretion of the gastric juice, whereby the food was prevented from undergoing those various chemical changes which are indispensable for proper solution and absorption. It is now known, however, that there are many chronic forms of indigestion which depend, not upon any *failure* of the gastric functions, but rather upon an abnormally active or excessive secretion of the gastric juice, the pain, acidity and vomiting which occur during the process of food-digestion being the result of *violent irritation of the mucous membrane of the stomach by its own secretion.* It is also probable that the majority of the cases which are habitually diagnosed as "gastralgia" arise in a similar manner.

It has, therefore, become necessary to renounce many of the superstitions which were formerly current concerning the ætiology of functional disorders of the stomach, and to substitute certain chemical methods of diagnosis for those which afford more equivocal results. In view of the practical importance of the subject, I shall venture to direct your attention from time to time to some of these less studied varieties of indigestion, pointing out the chief characteristic features they possess, and the line of treatment which is most usually successful.

Hyperacidity. By this term is understood a variety of dyspepsia which is constantly associated with presence of a gastric juice containing an abnormal amount of free hydrochloric acid; the secretion only occurring as a result of the ingestion of food, and ceasing as soon as the process of digestion is complete.

Sketch of a case.—

A young adult who has hitherto perhaps enjoyed excellent health commences to experience a sense of discomfort at the epigastrium after meals. The appetite remains good, the general health unimpaired, and there is neither nausea nor vomiting. By degrees, however, the uneasy sensation increases, and finally assumes the character of actual pain. Sometimes it is the breakfast, at other times the mid-day or evening meal which seems to disagree, or each in turn may be followed by unpleasant symptoms. Under the impression that the disease is only of a temporary nature and will soon wear itself away, the patient usually has recourse to such simple remedies as he may be able to command, or his friends recommend, until at last, wearied by constant failure to rid himself of his complaint, he seeks medical advice. The description which he now gives of his symptoms is generally sufficiently characteristic to afford an instant clue as to the nature of the malady. The pain does not ensue immediately after a meal, but its advent is postponed for a period of time varying from $1\frac{1}{2}$ to 3 hours. It commences as a hot or scalding sensation in the epigastric or umbilical region, and gradually acquiring a cutting or stabbing character, diffuses itself over the neighbouring regions of the abdomen and the lower part of the chest. Sometimes it is the hepatic region which is particularly affected, and this, coupled with the extreme severity of the pain, may lead to the belief that a gall-stone is responsible for the symptoms. Very often an intense burning sensation is experienced behind the centre of the sternum, and mouthfuls of an acid fluid are regurgitated, which scald the throat and set the teeth "on edge." But however severe the pain may prove, the patient always finds that he can obtain relief by having recourse to food or drink, while large doses of bicarbonate of sodium act even more efficiently. In most instances, however, the relief is only of a temporary character, and after a short interval, the pain returns with increased

virulence. As a rule, the paroxysm subsides spontaneously after an hour or two, but the patient will often prefer to cut the attack short by pushing his fingers down the throat, and causing the stomach to eject its noxious contents. This series of symptoms may be repeated after each meal, and the patient suffer from three distinct attacks during the course of each twenty-four hours; but more commonly it is only the principal meal of the day which gives rise to pain, and hence the attack occurs either about 4 o'clock in the afternoon, or at 10 o'clock at night.

At first the symptoms are intermittent in their appearance, and may fail to recur for many days or for even many weeks at a time; but these intervals gradually become shorter, until the attacks are of daily occurrence. The bowels are almost always confined, but in the later stages of the complaint diarrhœa is apt to supervene, owing to catarrh of the colon. The appetite is maintained throughout the whole course of the disorder, and the patient often exhibits an inordinate desire for food within a short time of a full meal. Extreme thirst is a constant symptom of the case, and the buccal cavity is often dry and the breath sour-smelling, owing to a deficient secretion of saliva. The tongue presents a clean, red, and moist appearance, and is seldom furred or indented along its margin by the teeth. The urine is neutral or alkaline in reaction, and is often turbid from the presence of phosphates. If an examination of the abdomen is undertaken during the height of the paroxysm, the superficial muscles are found to be in a state of rigid contraction. Manipulation of the epigastric and umbilical regions is productive of pain, especially when pressure is made over the region of the pylorus. No signs of dilatation of the stomach can be detected.

From this brief sketch it is obvious that we have to deal with a variety of dyspepsia which possesses three peculiar features :—

1. The violent pain which constitutes the characteristic symptom of the disorder does not occur until a late period of digestion, i.e., from two to three hours after the meal.

2. It is temporarily relieved by the ingestion of nitrogenous food, but more effectually by large doses of alkaline salts.

3. Frequent regurgitations of an acid fluid take place, but nausea and vomiting are rarely encountered.

How are we to explain these various symptoms?

The answer is by instituting a chemical examination of the contents of the stomach. If a patient suffering from this form of dyspepsia be given a light meal in the early morning, consisting of a dry roll and a pint of weak tea, and the contents of the stomach extracted an hour later, the semi-digested material is found to exhibit the following features. It possesses no unpleasant smell, and filters readily through paper, while the fragments of food which remain upon the filter present the swollen and gelatinous appearance that results from exposure to an active gastric juice. The filtrate is a limpid and slightly opalescent fluid of a pale yellow colour, which is capable of being exposed to the air for a length of time without undergoing decomposition. With litmus paper it exhibits a strongly acid reaction, but it contains *no lactic acid*. The presence of *free hydrochloric acid*, on the other hand, is easily demonstrated by means of Congo red paper or phloroglucine vanillin, while quantitative analysis proves that the amount of mineral acid is vastly in excess of the normal, varying from .3 per cent. to .7 per cent. The filtered fluid is also found to be endowed with extremely active digestive properties, thus indicating that the amount of pepsine is also in excess of the normal ; while from the fact that after neutralization with soda the filtrate is still capable of coagulating milk, it is reasonable to infer that the rennet ferment is in no wise deficient. An examination of the sour fluid which regurgitates during digestion, and of the vomit when emesis occurs, gives exactly similar results. Finally, it can be shown by the aid of the stomach tube that gastric digestion is completed and the stomach consequently emptied much more speedily than under normal conditions, and also that during the intervals of digestion no secretion of the gastric juice occurs.

With these facts before us it is not a difficult matter to assign the various symptoms of the disorder to their respective causes. Modern physiology teaches that in a healthy stomach the proportion of hydrochloric acid in the gastric juice does not exceed .2 per cent. and that with this degree of acidity the digestive functions are carried out in an orderly and entirely painless manner. If, however, the contents of the stomach acquire an abnormal degree of acidity, the sensory nerves of the mucous membrane at once become irritated, and pain results. It is also known that the presence of *free* hydrochloric acid cannot be

demonstrated in the contents of the organ until after the lapse of one or two hours from the termination of the meal, the earlier portions of the secretion being monopolized by the albuminous constituents of the food and retained by them in the form of a chemical combination which is digestively inert. And since it is only the *free* acid which is capable of exerting an injurious influence upon the mucous membrane, it follows that the resultant pain will not make itself felt until at a late period of digestion, *i.e.*, one to three hours after the meal. The chemical affinity which thus exists between albuminous substances and free hydrochloric acid affords an adequate explanation of the temporary relief that follows the ingestion of nitrogenous food. The arrival in the stomach of a fresh supply of proteid material serves to saturate a portion of the *free* acid and to render it incapable of causing further irritation. The relief, however, is only temporary, since the introduction of the extra food causes a fresh secretion of gastric juice to take place, and eventually the pain returns with increased violence. Large draughts of water prove beneficial by mechanically diluting the hyperacid secretion, while the administration of alkalies is productive of more permanent benefit by effecting the neutralization of a considerable portion of the offending acid.

Another result of this chemical irritation of the stomach is to induce an excessive amount of peristaltic movement in the organ, which, by hurrying the food into the intestine, is possibly responsible for the sensation of hunger which recurs so soon after the meals. The obstinate constipation which usually accompanies this form of dyspepsia appears to depend chiefly upon the neutralization of the bile and pancreatic secretions by the abnormally acid contents of the stomach.

The exciting causes of hyperacidity are numerous and apparently diverse, but they all probably exert their specific action through the medium of the nervous system. Thus, sudden and severe emotions, such as anxiety and fright, have been known to be followed within a short period of time by this form of indigestion; while prolonged mental or intellectual strain not infrequently terminates in a similar condition. Certain affections of the brain or cord, both functional and organic, are apt to be accompanied by an excessive secretion of the hydrochloric acid, notably hysteria, neurasthenia, and sclerosis (ataxia). Occasionally the gastric disorder arises during the convalescent period of some specific fever, and at the present moment I have a well-marked example of the complaint under my care, in which the first symptoms followed immediately upon an attack of malaria. More commonly, however, the disease springs directly from a prolonged and excessive stimulation of the mucous membrane of the stomach. Persons who habitually indulge in copious meals consisting largely of nitrogenous material, educate their stomachs to pour forth an abundant secretion of digestive fluid upon each occasion. Accordingly, so long as the daily consumption of food continues to be excessive, the gastric secretion does not exceed in quantity that which is required to dissolve the nutrient ingesta, and, consequently, no untoward effects are observed.

But should the diet be restricted from some cause or another, the stomach still continues to secrete an amount of juice which proves to be in excess of its immediate needs, and consequently the symptoms of hyperacidity manifest themselves. This is probably the reason why so many *bon vivants* become affected with acid dyspepsia when circumstances have necessitated their observance of a strict *régime*. Highly spiced foods and condiments may also give rise to hyperacidity from the stimulating influence which they exert upon the mucous membrane of the stomach, and a similar result is apt to follow the unrestricted indulgence in such things as beer, tea, coffee, spirits, and ices. Finally, an excessive secretion of the gastric acid may ensue as a direct result of some long-continued local irritation, such as chronic ulceration of the inner surface of the organ.

The clinical course pursued by cases of hyperacid dyspepsia presents considerable variation. In most instances the disorder is markedly intermittent at its commencement, and when properly treated sometimes fails to recur. In the majority, however, relapses become more and more frequent until at length the complaint assumes a chronic character, and a paroxysm occurs at least once a day. In very chronic cases the stomach may exhibit a kind of paralytic secretion, the production of the gastric juice being no longer dependent upon the ingestion of food, but occurring spontaneously in the empty organ. In this manner a case of hyperacidity becomes gradually converted into a much more severe disease known as hypersecretion, with the distinctive symptoms of which I hope to deal in my next lecture.

Hyperacid dyspepsia may require to be diagnosed from four other diseases, namely, *gastric ulcer, biliary colic, hypersecretion* and *gastralgia.*

From *gastric ulceration* it may usually be distinguished by the fact that in the latter disease the pain which results from the ingestion of food occurs shortly after the meal, and is seldom deferred for two or three hours. It is also more localized, and generally terminates in vomiting. In both cases, however, the contents of the stomach may be found to contain an excess of free acid.

In its general features, *biliary colic* may closely resemble those of hyperacidity. The hepatic complaint, however, is much more common in women than men, the paroxysm fails to exhibit any definite relation to the meals, and the pain while it lasts is much more severe and is not relieved by food. After the subsidence of the paroxysm a correct diagnosis is easily arrived at.

From *hypersecretion* the disorder can easily be distinguished when recourse is made to the stomach tube, the presence of gastric juice in an empty stomach being pathognomonic of the former. Vomiting and dilatation of the stomach are also common in hypersecretion, but rare in hyperacidity.

In true *gastralgia* the pain is often irregular in its onset and usually appears independently of the meals, while the composition of the gastric secretion presents no deviation from the normal. It is exceedingly probable, however, that many cases which are diagnosed as gastric neuralgia are, in reality, instances of hyperacid dyspepsia.

Treatment. The general treatment of this disorder does not differ materially from that of other forms of indigestion. The patient should be warmly clad, take a moderate degree of exercise, and observe regular hours. The use of tepid salt baths or douches to the epigastrium have been highly recommended, while a flannel belt or bandage worn round the upper part of the abdomen is sometimes of considerable service.

In every case the *diet* must be carefully regulated. The peculiar property possessed by proteid food of being able to combine with free hydrochloric acid renders the employment of nitrogenous materials of the greatest importance. Meat of all kinds, as well as fish and game, may be allowed, but they should be minced as finely as possible, and carefully masticated before being swallowed. Milk and eggs are particularly valuable, and are best administered in small quantities at short intervals. Vegetables, and especially those containing an excess of starch, must be avoided as far as possible, since they call forth a plenteous secretion of gastric juice without being able to control its acidity. All spices, condiments and curries, as well as tea, coffee, wine, and liqueurs, must be prohibited. If some form of stimulant is considered imperative, a small quantity of good whisky, well diluted, may be allowed. To allay the thirst and also with a view of relieving the pain, the patient may be encouraged to indulge freely in milk, milk and seltzer, lithia or soda water, or in any of the natural or artificial alkaline waters. The relief of the pain constitutes the first and most important indication in the employment of *drugs.* With this object recourse must be made to such alkaline salts as the bicarbonates of sodium and potassium, calcined magnesia or the solution of potash. Of these I prefer the first named, and am in the habit of commencing the treatment by prescribing it in doses of 30 to 60 grains, dissolved in two ounces of water.

The patient is directed to take the first dose two hours after each of the principal meals, and to continue every hour till four doses have been taken. Under this treatment the pain after food rapidly diminishes and finally subsides, pyrosis disappears, and the bowels are moved freely. Continental writers also advise that the patient be placed upon a course of sedatives, such as bromide of potassium, atropine, or opium, with the object of subduing the abnormal excitability of the gastric nerves. I have, however, found it a better plan to maintain the alkaline treatment for a considerable length of time, small doses of the liquor potassæ combined with the bromide being administered twice a day between the meals; while a pill containing podophyllin, creasote, colocynth, and rhubarb is prescribed each night. If the state of the bowels demands active supervision, simple purgatives or enemata will be found to be most serviceable.

THE

ELECTRO-THERMO-CAUSTIC LOOP.

By W. S. HEDLEY, M.D.

THE employment of the electro-thermo cautery is often disappointing. It is apt to fail at the most critical moments. Especially open to this objection is the electrically-heated loop or noose. It may be said often to prove *a delusion* as well as

a snare. Such an experience as the following is not quite exceptional. An operation by the method in question is decided upon. The cautery battery in working up to its full output, the connecting wires are properly attached, the resistance is placed in circuit, and, the current being turned on, the wire is brought to the required glow. Satisfied with his experiment, the operator adjusts the loop, tightens it a little, and, with a light heart and a firm hand, presses the contact that completes the circuit. The wire reddens; but no sooner does it sink into the tissues than its glow disappears and its progress is arrested. The operation is postponed, or finished by some cutting instrument, or possibly by some electrolytic procedure hastily devised. The operator, ignorant, perhaps, of electro-physics, bitterly blames the method, and may abjure its employment for ever; or possibly it may occur to him in some measure to blame himself, and patiently to inquire into the causes of his failure. It is, no doubt, largely due to such disappointments that the methods in question have to a great extent been abandoned. But contributory to such a result there are also considerations of another kind. Voltolini's objection to the use of the galvano-caustic loop in the ablation of hypertrophied tonsils seems to have been that the wire was so slow in making its way through the sometimes dense tissues that a dangerous amount of heat radiated to neighbouring structures. It cannot be said that any of these objections are without foundation; but with modern improvements in instrumentation they clearly lose much of their force. They seem to be traceable to three causes (1) imperfect means of electrical supply, (2) want of accurate measuring instruments, (3) the use of a material for the loop, which is not the most suitable. The first of these drawbacks is attributable to the fact that the electromotive force of the average cautery battery is far from constant, and that the output of current is very uncertain and soon exhausted. The phenomena of "polarisation" in a great measure explain this, inasmuch as a "back" or counter electromotive force is thereby produced, which, acting as a resistance, tends to cut down the voltage of the battery. The modern accumulator, however, without polarisation, with a low internal resistance, and an electromotive force which is practically constant at two volts, presents none of the above disadvantages. Given, however, a satisfactory supply of current, there still remains

the necessity for measuring it. An ammeter certainly, and a volt-meter, if available, ought always to be at hand.* As to the third of the above points, it is experimentally determined that, in comparison with platinum, a steel wire requires a less current strength (intensity) to bring it to a given temperature. A steel wire cautery loop $\frac{1}{4}$ mm. in diam., requires 5 ampères to carry it to dull redness, 6 to redness, $6\frac{1}{2}$ to cherry redness, $7\frac{1}{4}$ to white heat, 8 to fusion (Cheval). How does this help to an explanation of the operative fiasco above referred to? Let an experiment help to answer the question. Take twelve inches of steel wire, and, passing a current through it, bring it to redness. Then, with the current still running, let a stream of water play upon a portion of the heated wire. It will be found that the wire on either side of the cool part becomes hotter than before.† The converse can be shown by substituting the flame of a spirit lamp for the stream of water.

These two experiments illustrate the fact that the electric resistance of conductors is a function of temperature. The resistance of a wire increases with its temperature, and the resistance of a steel wire increases in a greater ratio than does that of a platinum one. Now in view of the fact that the principle of electric heating is the transformation of electrical energy into heat by overcoming resistance, it follows (1) that steel wire having a higher resistance than platinum as well as a higher ratio of increase in resistance when heated, will so far be a better material for the caustic loop. (2) That when the heated wire passes into the tissues it becomes by its lowered temperature a better conductor, i.e., its resistance diminishes; there is less work to be done in overcoming that resistance, less electrical energy, therefore, is converted into heat at that point, and the current expends its power on other parts of the circuit. It must not be assumed that because a certain current (say 5 ampères) is sufficient to heat to the required redness a wire of a certain diameter, say $\frac{1}{4}$ mm., in air, that therefore 5 ampères is the proper operating current. To maintain the proper glow in passing through the tissues a higher intensity (say 8 ampères) will be required.

The electro-thermo cautery adapts itself to the requirements of minor surgery in many well known ways. Its varied uses in laryngology and rhinology

* These readings once determined for any particular apparatus, it is, of course, unnecessary to verify them for every separate operation. † Byrne.

may be instanced, its suitability for the removal of pedunculated or other tumours, for the treatment of fistula and hæmorrhoids, for the cauterisation of pedicles, even for the removal of some cancerous growths. The ablation of hypertrophied tonsils may perhaps be regarded as the special *metier* of the electro-thermo-caustic loop. Yet, although it is forty years since Middeldorpf advocated its use, it at this day finds itself laid aside in favour of methods more dangerous and less effective. There are preferred before it the bistoury scissors or amygdalotome with no little attendant risk of hæmorrhage, ignipuncture with its slowness and post-operative dangers, sometimes even electrolysis, with its frequent repetitions and doubtful results. To restore the cautery loop to surgical favour there only seem to be required a well arranged instrumentation, and a source of electrical supply which will, without fail, and without much trouble to the operator, meet the demands that may be made upon it. In other words with accumulators of sufficient output* and a commercial current at hand ready to recharge them, with a steel wire of ¼ mm. for the loop, with a rheostat and an ampère-meter (the latter measuring up to ten ampères), with the wire carriers strong and well insulated, with a suitable handle for constriction and contact, conducting cords of sufficient capacity, the invariably limiting the operation to one tonsil at a time, and the not forgetting the precaution of ice-swallowing for a day or two after the operation, —the risk of failure will be slight, and post-operative troubles almost always insignificant.

LECTURES

ON

THE METHODICAL DIAGNOSIS OF DISEASES CAUSING DEAFNESS WITHOUT PAIN OR DISCHARGE.

Delivered at the Central London Throat and Ear Hospital, Grays Inn Road, Jan. 8, 1895,

By DUNDAS GRANT, M.D.,

Physician to the Hospital.

I.

GENTLEMEN,—With regard to the subject of to-day's lecture, viz.: the methodical diagnosis of diseases of the ear causing deafness, I shall premise, in the first instance, that patients suffering from disease of the ear come to us complaining of certain cardinal symptoms, either defective hearing power, pain, discharge from the ear, noises in the head, giddiness, or visible alterations in the organs. I propose to devote a lecture to the diagnosis of the cases under each of these headings. To-day I shall confine myself to the class of case in which patients complain of defective hearing without pain or discharge. Now, in the first place, we have to determine whether the case is one of "obstructive" or of "nerve" deafness, for we subdivide cases of deafness into two classes,—the former arising in the external and middle ear, the latter in the internal ear or the central continuations of the nerve of hearing. The classes are fairly distinct.

I shall not go through the various tests of hearing, because I think they must be familiar to most of you, but as a great deal centres on the test of hearing by bone conduction, I shall just remind you that our first point of distinction depends on whether "bone-conduction" is increased or diminished. Now, it may be said, as a general rule in simple obstructive deafness, that bone-conduction is increased or is normal; it is not diminished. In nerve-deafness the bone-conduction is diminished. That is the first great fact. (Dr. Grant exhibited a patient with nerve-deafness. When the patient had ceased to hear the tuning fork applied to the bone of the ear, others heard it quite distinctly). It should be heard for a definite time dependent on the stroke of the tuning-fork, but I shall not go into details on that subject to-day. The next is what is called Rinné's test. Now, if a tuning-fork in vibration is placed in contact with the bones of the skull until the sound dies away, and then held opposite the meatus, it is heard by the patient once more, that is to say, if the middle and external ear are in a normal condition, quite irrespective of the condition of the internal ear. When the fork is again heard in the way I show, Rinné is said to be "positive" and in the other case "negative."

Here is a case we have not seen before. She hears the fork longer on the bone than opposite the ear. In her case Rinné's test is negative, and it is obvious she has some obstructive deafness, whether or not she has nerve-deafness as well. Now, you see, she hears this fork after I have ceased to hear it. Her bone-conduction is, therefore, increased.

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* A battery that may be safely discharged up to, say, 10 ampères for short periods.

The next is Weber's test, and it means that if the tuning-fork is placed upon the vertex and one ear is obstructed, the sound is heard loudest or only in the obstructed ear. Then Weber is said to be "positive." You can test it very well by artificially obstructing the meatus. If, on the other hand, there is a defect in one of the auditory nerves itself, of course the tuning-fork on the vertex is not heard so well in the ear in which the nerve is affected, and Weber is then said to be "negative." I dwell upon these for the moment because so much turns on them from the point of view of diagnosis.

If, then, on testing with the tuning-fork, the bone-conduction is normal or increased, Rinné's test is negative and Weber's test is positive, we may diagnose at once obstructive deafness. If, on the other hand, we find bone-conduction is diminished, Rinné positive and Weber negative, that is to say, that the fork on the vertex is heard better in the good ear, we may diagnose nerve-deafness.

I would just mention one or two other points which would help to substantiate the diagnosis. In obstructive deafness, hearing power is better for high-pitched tones than low ones, as we can test by means of Galton's whistle. This is graduated according to the length of the tube. As I now blow it a tiny whistle is heard, and a patient who has merely obstructive deafness can hear the very high-pitched notes, because the tympanum has very little to do with the conduction of such sounds. Then another point: in subjects of obstructive deafness, hearing, as a rule, is worse in proportion for conversation than it is for the watch-tick. That is to say, in obstructive deafness the patient may hear the watch-tick a few inches away, and yet may be quite unable to hear conversation unless extremely loud. On the other hand, in nerve-deafness, a patient may be perfectly deaf to the watch, even on contact, and yet may be able to hear conversation relatively extremely well. Then, in obstructive deafness, if bilateral and severe, hearing is better in the midst of a noise.

Having got so far, Gentlemen, let us assume for the moment that a case is one of OBSTRUCTIVE DEAFNESS. We will follow a very natural and practical classification by dividing the diseases into the sudden, the acute, and the chronic; and this lends itself extremely well for diseases of the ear. Now, let us suppose the deafness (obstructive) is SUDDEN. The patient comes and tells you he has

suddenly become deaf, and on applying the tuning fork you find it is obstructive deafness. The probability is immensely in favour of it being *impacted cerumen*, but it would be most unwise to make the diagnosis without further inspection, although it would put you on the look-out. Now, supposing that it is ACUTE, that is to say, it has come on in a day or two days, leaving out of account those cases in which there is either pain or discharge, then we may almost certainly say the cause is *Eustachian catarrh*. Now, the means by which you confirm this diagnosis would be that your patient was suffering from cold; that on inflating with Politzer's bag, and listening with the diagnostic tube at the same time you heard that the Eustachian tubes were narrowed, and when the air traversed the Eustachian tube and entered the tympanum you found at once an enormous improvement in hearing. The only appearance you would have in the drum would be indrawing of the membrane.

Now, supposing that our obstructive deafness, instead of being acute, was CHRONIC, having come on gradually, or being of old standing. We next ask whether there has been any discharge from the ear. You have to insist on that question, which is very important. You should know, if possible, whether the disease arose with an acute catarrh or in an insidious way. If there has been no former discharge from the ear, you may say the case is one of chronic non-suppurative catarrh of the middle ear, of which there are two forms, the sclerotic and the exudative. In the latter there is implication of the Eustachian tube, which has generally arisen in naso-pharyngeal catarrh, and may be associated with adenoid growths in the pharynx. Part of the dulness of hearing is due to the indrawing of the membrane, owing to narrowing of the Eustachian tube. We therefore apply the test of inflation, and in such cases we find on auscultation that the tube is narrowed, and the air does not enter readily, but when it does enter it produces an improvement which takes a considerable time to disappear. In that case you have to deal with *exudative catarrh of the middle ear*, which is not of hopeless prognosis, and in such cases it has usually commenced with some definite catarrhal attack. Furthermore, tinnitus is not a constant or primary symptom. On the other hand, if it has come on very insidiously—the patient has hardly known how—and from the very commencement has been associated with almost constant

tinnitus aurium, then if you find on inflation that the Eustachian tube is not particularly narrowed, and that the active inflation produced little or no improvement, you have to do with *chronic proliferative catarrh* of the middle ear—a dry form of insidious sclerosis, which is the cause of much incurable deafness.

Just to return for a moment to the exudative catarrh, I mentioned that on inflation there was a certain amount, it might be a considerable amount of improvement, and if the Eustachian narrowing was not very great, and if the tympanum and tympanic membrane preserved some of their elasticity, that it might last some time—say several hours, a day, or a day and a half. If you find that after considerable improvement this improvement passes away almost at once, that is to say, almost at the next act of swallowing on the part of the patient, the probability is that the drum membrane has lost its elasticity, it has become relaxed. In such case the membrane is drawn in to the utmost extent that a membrane can possibly be. As shown in this diagram, it looks as if there was an entire loss of the tissue of the posterior half of the membrane, so that you can see the articulation of the incus and stapes, and the niche for the fenestra rotunda; but on inflation, the relaxed portion of the membrane is blown out in the manner shown. Now that is *relaxed membrane*, and the usual way in which it is tested is by placing Siegel's speculum in the ear and exercising suction with it, when you may see the membrane covering these parts drawn quite away from them; this is a frequent source of persistent deafness which is often overlooked.

Now suppose, on the other hand, the obstructive deafness has existed for some time, subsequent to a former considerable discharge, then it is due to the *results of suppurative catarrh of the middle ear*, which may leave a perforation or cicatrix. The easiest way to distinguish these is to inflate the ear, and if you can hear the characteristic sound you may say there is *perforation*. In such a condition as this you may see a well-marked hole in the membrane, through which the inner wall of the tympanum is distinctly visible. On the other hand, if there is no perforation sound, the certainty is that the membrane has healed up; and on inspecting it you may perhaps see a little depression such as this indicating the position of the *cicatrix*, where there has formerly been a perforation. Remember, however, you may sometimes see what

looks unmistakably like a perforation, and you think it must be one ; but on inflation you get, to your surprise, no perforation sound—the real condition being a thin indrawn cicatrix which may be more or less adherent.

Let us consider now the case of a patient who has diminution of bone-conduction, and the other signs of NERVE-DEAFNESS. Now nerve-deafness is a very large term, as you will see, and many points in its diagnosis are dependent on data of great uncertainty, requiring analysis in which the neurologist has really to come to the assistance of the aurist.

Let us suppose a case of nerve-deafness which has come on SUDDENLY. For the sake of completeness I have made a distinction between those occurring with injury and those without injury. If suddenly, without injury, nerve-deafness comes on with extreme vertigo, vomiting, noises in the head, and almost complete collapse, you have a well-marked picture of that not very uncommon affection known as Ménière's disease, which in this form is a *labyrinthine effusion*. This is the most important kind of sudden nerve-deafness with which you have to deal. (The questioning of a patient by Dr. Grant elicited the fact that he was sitting having his dinner when the deafness suddenly came on. He tried to get up, but fell back again. He had no noise in his head whatever, but afterwards had a dull noise in his ear, which still continued.) In some cases it simulates a bilious attack ; so much so, that people will tell you the deafness came on suddenly, with an attack of congestion of the liver. That has occurred in my experience several times ; and of course it is perhaps easier to diagnose afterwards when the gastric symptoms have disappeared.

Now, we come to a form of ACUTE nerve-deafness. You have a case of extreme nerve-deafness which has come on within a very few hours—a day or a day and a half, perhaps—you would call that acute. Say there has been no pyrexia, there is no evidence of congestion of the brain nor of anæmia. This form of nerve-deafness is usually due to *syphilis of the labyrinth*. It may come on in the secondary stage or in the tertiary, and, what is the most distressing form, in the subjects of hereditary syphilis. In these it occurs about puberty and is preceded by interstitial keratitis, accompanied by the characteristic Hutchinson's teeth.

In a case of CHRONIC nerve-deafness, you

should search whether there is any further implication of the nervous system or not. For the moment I am going to point out to you the chief types of chronic nerve-deafness occurring without further implication of the nervous system. You find, on inquiry, that the patient has been habitually exposed to noises. You have then what is called *boiler-maker's deafness*, because it is most severe in people following the business of boiler-making, especially those who work inside while the hammering is going on. It often happens in artillerymen. Frequently patients with chronic nervous deafness have been exposed to noises when you would not have expected it. For instance, I had the case of a lady with typical nerve-deafness in one ear. I found that she habitually sat in the neighbourhood of wood-carving and wood-chipping. The ear which was nearest to the noise had become markedly affected. Then another class of patients consists of gamekeepers or other shooting men. In these the most affected ear is almost invariably the left one, on account of the position of the fowling-piece. Again, you have the *toxic* form, which comes on in people who have to take large quantities of quinine.

Then you may have nerve-deafness with implication of other cranial nerves; for instance, with paralysis of the facial nerve. If this occurs in the subject of otorrhœa, the two nerves are probably affected where they lie together in the labyrinth. This is usually due to tubercular suppurative *disease of the petrous bone*. If the auditory and facial nerves are paralyzed in a case where there has been no suppuration, there is almost certainly disease affecting the *intracranial portion* of the auditory nerve. If there is paralysis of the sixth nerve, it is in those cases where the affection of the eighth nerve has been due to *basal meningitis*—not at all an uncommon thing in the previous history of some of our cases. On the other hand, it occasionally happens that the spinal accessory nerve is affected, as shown by paralysis of the vocal cord. You have sometimes to look a considerable distance off to get confirmation of the cause of nerve-deafness. Now if you have paralysis of the spinal accessory nerve with the auditory nerve, the most likely place for it to be is external to the medulla oblongata, and would then most probably be thickening of the meninges of a syphilitic character, or a new growth situated on the posterior aspect of the petrous bone near the apex. On

the other hand, we may have implication of the central nervous system. Supposing along with the nerve-deafness we have simultaneously hemiplegia, we naturally assume that the disease causing hemiplegia at the same time affects the opposite *temporo-sphenoidal lobe* or the strands leading to it. One is apt to overlook "word-deafness." The patient can hear all sounds, he may tell you you are talking quite loud, but he cannot understand a word you say. That is due to disease of the *left temporo-sphenoidal lobe*. If, on the other hand, there is locomotor ataxy, then in the later stages of that disease you sometimes have degeneration of the auditory nerve or nuclei, to which you would attribute the deafness. In disease of the *cerebellum* the symptom of reeling about may be associated with deafness, but this is not the rule.

I will now refer to the one or two cases I jumped over. You may have a nerve-deafness with cross-paralysis. That would occur in the *pons*, and naturally would be almost certainly bilateral. Hemianæsthesia coming along with nerve-deafness would indicate that the trouble was at the posterior part of the *internal capsule*. Suppose the nerve-deafness was due to injury—a blow on the head—producing sudden nerve-deafness, then it is probably a *concussion* or *fracture*. The probability is there was concussion of the labyrinth, unless there was distinct evidence of fracture to the base of the skull, when it would probably be a *fracture of the capsule of the labyrinth*. With regard to acute cases I have already mentioned *syphilis*, apparently with very little other symptom except the onset of the deafness. There is also a form of nerve-deafness attributable to *congestion of the labyrinth* connected with what would be called congestion of the brain, and it would be naturally associated with very considerable vertigo and noises in the head, and these noises in the head would probably be of a pulsating character. There would be other evidence, such as headache, sleeplessness, and so forth. On the other hand, deafness may be associated with *anæmia of the labyrinth*, as from extreme loss of blood, parturition, etc., improved on lying down.

We have another class of cases which almost invariably elude detection at the time of the onset. A patient is extremely ill, with high fever, and symptoms almost unmistakably those of meningitis, when of course during the period of insensibility the hearing power cannot be tested. It is

found that the meningitic symptoms disappear with a rapidity which is quite unusual in that disease, and leaves behind absolute deafness. This is due to *inflammation of the labyrinth*—labyrinthitis. In the cases in which it is not preceded by unmistakeable meningitis, and has been caused neither by infectious disease nor by injury, we can only call it *simple labyrinthitis*. If it has been associated with infectious disease—scarlet fever, mumps, etc.— we must call it *infective labyrinthitis*, and that is not a very infrequent condition, though of course the affection of the labyrinth is eclipsed by the severity of the general disease.

Again, it may follow injury ; that is to say, there has been an injury to the head, and some few days afterwards signs of fever and nerve-deafness. Then you have to do with *traumatic labyrinthitis*.

In other cases, you may have inflammation of the labyrinth preceded by decided meningitis ; that is to say, the meningitis runs its ordinary course, and after a protracted illness the patient may get well, but remain deaf, with no other implication of cranial nerves. There is then no doubt it has been an extension of inflammation from the meninges to the interior of the labyrinth, which, as you know, may take place through the lymphatic channels—*meningitic labyrinthitis*.

Functional deafness may arise from mental shock, from *hysteria*, or from *angioneurotic* disturbance, or it may be *simulated*. These puzzling cases can only be cleared up by an analysis of the general conditions, due attention being given to such points as abnormal emotional disposition, sudden alternate flushing and pallor, desire to obtain damages, or to escape military service, or to leave school, such as will readily be appreciated by the practitioner if prepared for their occurrence.

So much for pure obstructive deafness and pure nerve-deafness. Naturally there is a possibility of combination of the two, and if we find bone-conduction is diminished and Rinné is negative, then we have, as in the young man I showed you just now, a COMBINATION of NERVE and OBSTRUCTIVE DEAFNESS.

Suppose you have established that condition, you have to apply the old principles of diagnosis, but the following is probably the quickest way :— We find out whether there has been a previous discharge. If there is no history of such obtainable, you ask, is the case primarily one of chronic disease of the middle ear ? You investigate whether there was an insidiously increasing deafness from the

very commencement, with probably better hearing in the midst of a noise. Very slowly it has got worse, but all at once it takes on a much more rapid progress, and we have extreme increase of deafness, to such an extent indeed, that the patient can only hear a loud shout. Then it may be due to either secondary implication of the labyrinth ; or to the stapes having become fixed in the fenestra ovalis. If the diminution of bone-conduction is very slight there is probably *ankylosis of the stapes ;* if considerable, we may be certain that there is secondary *affection of the auditory nerve*, and probably in the first turn of the cochlea.

Now, on the other hand, supposing that we do not elicit a history of chronic catarrh and primary nerve deafness, what would lead you to suspect primary nerve-deafness ? It would be the fact that the patient was perhaps exposed to noise, or that it had come on suddenly with the symptoms of Ménière's disease, with no precedent deafness, or it may be that the patient is the subject of hereditary syphilis, and so shows all the signs of that cachexia, and has got much deafer recently ; or it may be the patient could tell you he knew he always heard worse in the midst of noise. There is no reason why you should not have superadded catarrh of the middle ear. That is a point to be kept well in mind before dismissing a patient with syphilis of the internal ear, without examining the middle one carefully.

Now, suppose on the other hand, we have a history of considerable discharge from the ear, and that we have superadded to it the signs of nerve-deafness ; then we may say that *the suppurative disease has extended from the middle ear to the labyrinth*. This is not at all an uncommon sequence of events, but it usually occurs in tuberculous cases.

Before I conclude, there is one caution I would like to give you with regard to Rinné's test, which I think is so important, and I myself have made so many mistakes from want of knowledge and appreciation of it, that I feel bound to warn you all against it. It is this :—In a case of unilateral nerve-deafness, the other ear being perfectly good, I put my tuning-fork on the mastoid of the deaf side, and it is heard ; when it ceases to be audible, I place it opposite the meatus—the patient does not hear it again. This is " negative " Rinné ; and you say " negative Rinné—this is obstructive deafness." The point is this :—that the sound is conveyed through the bones to the

opposite ear, and the bone-conduction is evidently derived from the healthy ear. The error is checked by the employment of Weber's test, which in such a case is "negative."

(Dr. Grant then showed a case in which the fork applied to the bone of the deaf ear was heard in the good one. Weber in that case is negative, pointing to nerve-deafness. After further tests, Dr. Grant said that looked like a negative Rinné, but if he were to treat the case for chronic catarrh in the middle ear, he would be quite at sea, because negative Rinnè must be supported by positive Weber. This illustrated the fallacy in which he trusted none of his audience would fall.)

Here is a case of extreme nerve-deafness, with the singular feature that the nerve-deafness came on six months after severe injury to the head. I cannot offer an exact explanation of it. But it may be that "callus" or some thickening of the meninges of an inflammatory form has gradually strangled the auditory nerve. (Case shown).

(In conclusion, Dr. Grant begged his hearers not to trust solely to this diagnostic scheme, but to use it as a sort of guiding thread to lead them through the intricacies of their cases and of the voluminous works to which he hoped they would refer with greater ease and profit from having utilised this methodical system.)

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Tuesday, February 19, 1895.

Infective Angioma on the Hand. Nævus-Lupus.

THE subject of this case was a girl, aged 21, of whose hand two portraits had been taken some years ago, which are now to be seen in the museum. The original condition in early childhood was that of two nævoid patches in the skin of the back of the hand, between the thumb and forefinger. They were about the size of two sixpences, and were placed near together, with a third about the size of a pea close to them. Although certainly nævoid in part they were not purely so, for their surface was rough, and had a dry adherent scale crust. They had originated in early childhood,

and had not been present at birth. The disease was clearly aggressive and infective, and two of the patches were no doubt satellites to the other. The first portrait showed very accurately the condition of things in 1884. A second portrait taken in 1887 showed a recurrence of the nævoid condition in the scar which had remained after a very efficient cauterisation, which had been performed in the hope of cure. Although the scar was sound it was stippled over with tufts of dilated blood-vessels, almost black in colour, exactly like, excepting in point of tint, the vascular scars left after lupus on the face and scalp. At the present time the girl's hand showed a condition of acute inflammation around the patches which had occurred in connection with the recent cold weather, and a slight bruise which had been received. The patches had also increased considerably in size. The girl had no skin disease elsewhere.

Comments. I am very glad to have the opportunity of showing this patient, and of illustrating the persistence of the morbid process. It is a case of which I never saw the exact parallel. There are none of the dilated lymph-vesicles which characterise the more common examples of infective lymph-angioma. In the latter disease, however, there are always present some tufts of dilated capillaries, almost black in tint, and exactly like those which here constitute the whole of the disease. From the first I always regarded this case as a peculiar form of lupus. By others it was considered to be nævus. I admit that it was nævoid, *i.e.*, it was vascular and spongy, and could be emptied by pressure; but there was something more than that, *it was infective*. It spread at its edges, and it produced satellites. I appeal to its present condition, eleven years since the portrait was taken, and eight since the attempt to cure it by cauterisation, as affording some confirmation of my diagnosis. You see that the patches have enlarged, and that they are now passing through a condition of acute inflammation, just such as many of our lupus cases have experienced from the influence of the recent cold weather. The condition of the vascular scar that was left after cauterisation, a very peculiar one and exactly resembling what I have repeatedly demonstrated as occurring in some cases of lupus, gives also, I think, strong support to my diagnosis. Whatever you like to call the disease two points are certain. It is a very chronic infective malady, which extends slowly, and it is exactly the same as the

vascular part of what we know as lymph-angioma. I have no doubt that the infective element in the latter is the same as that present in this girl's hand, and I think it exceedingly probable that in both it is in association with the tubercle bacillus. I am aware that such a suggestion may seem to have but little foundation; but you must remember that there are many links in the chain, and that this conclusion is arrived at by the endeavour to keep them all in due connection. After all the present condition of the girl's hand is not much unlike that of a nævus upon which lupus necrogenicus has been engrafted.

An Example of Infective Angioma which had spread extensively over the limbs.

The subject of this case was a girl, aged 15, who was under the care of Mr. Waren Tay, at the Blackfriars hospital for skin diseases. She had first a long, broadish streak of dilated capillaries extending down the left forearm on its ulnar side, from elbow to wrist. This, she said, had been present five or six years, and had given her but little trouble. During the last few months, however, a similar condition had developed itself over a large portion of the left lower extremity from hip to ankle. On this limb almost the whole surface was more or less involved in what looked at first sight somewhat like a slight form of port-wine-stain, but which was more or less in streaks and patches, and which was attended by very minute lichenoid developments, which were polished on the top. The girl said that her cheeks, and especially the left one, were becoming affected by the same disease. To the observer it might have seemed that she was simply very florid, but on careful inspection it was clear that she was correct, for there were little tufts of dilated capillaries not only on her cheeks but down the side of the neck, and they were attended by some definite though very minute scars. Everywhere the dilated capillaries could be emptied by pressure, but not without difficulty, and they filled again instantly. In the case of the lower extremity, as in the forearm, a long streak at the back of the limb ran from the ischiatic tuberosity to the ankle. One other point has to be mentioned. On the back of the girl's right forearm there was a deep scar, almost adherent to the bone, which had remained after a strumous abscess in early childhood. She believed that a piece of bone had come away. She showed

no signs of inherited syphilis, and appeared to be in fair health.

Comments. We have in this case an example of a very superficial infective inflammation, spreading probably in the peri-vascular spaces, and showing its effects almost exclusively on the blood vessels themselves. It has produced no crusts, nor has anything attended it which, in the popular sense of the word, would be called inflammatory. The changes are rather like those of a very slightly developed form of nævus; but we must remember that they have shown a remarkable aptitude for travelling and have spread over a very large extent of surface, obviously by a process of contagion by continuity. They have also produced minute cicatrices, which, though very slightly marked and easily overlooked, are yet, I assert, quite definite in several places. Our Museum contains several portraits illustrating this disease, but in most of the cases, so far as they were observed, the tendency to spread was very slow, and was throughout limited to the limb or part first affected. In one of them, however, it covered almost the whole of one lower extremity of a young girl. In that case I used the descriptive epithet, "cayenne-pepper grains," for the limb looked as if it had been tattooed all over with minute grains of cayenne. I think you will admit that that expression is not wholly inappropriate to some of the appearances in our present patient, though by no means to all of them. The cases are of the same nature, but they are alike with differences. I have seen but one patient whose case goes on all fours with the present one, and that was a girl whom we had before us in this room only two months ago. She had been sent to us by Mr. David Walsh, and she also was one of Mr. Tay's patients. In her the condition was said to have begun a year before we saw her, and had gradually spread over the whole body and limbs. She was mottled all over by port-wine-stains arranged very irregularly, but running in some parts more or less in lines. When the blood was pressed out the skin appeared, at any rate in parts, to be thin and atrophic, and it was certain that, on the breast especially, there were numerous little scars. At the back of the left knee there was a large red discoloured patch which was crossed by some dilated veins. The girl was 18 years of age, and had suffered from rheumatic fever. It was also said that at the age of 7 she had spat up blood during an attack of bronchitis. I have had a portrait taken of this

girl's arm, but the conditions were too indefinite to lend themselves well to the artist's pencil. Mr. Walsh was inclined to use the term "atrophodermia" in reference to his patient. My own view of the case is that the atrophy was secondary to an infective inflammation allied to lupus. These two cases are exactly alike, differing only in the extent of surface involved in the changes. The affection of the popliteal space in particular in the two girls was precisely similar. You will see that in both we have some little ground for the supposition that struma, i.e., tuberculosis, existed. One patient had spat blood and the other had a large scar of a scrofulous abscess. Resting my diagnosis, therefore, chiefly on their infective character and scar-leaving tendencies, I feel justified in claiming the disease as a form of lupus. That it is neither lupus vulgaris nor lupus erythematosus I fully admit. The cases must make a separate group for themselves, and I know no better terms for them than infective angioma or nævus-lupus.

Before leaving these cases I must ask your attention to another feature which is well displayed in the first of them, but which has probably nothing whatever to do with the essential nature of the disease. I allude to the development of the changes in long streaks running lengthways down the limbs. These streaks are observed in several different maladies, notably, in ichthyosis herpetiformis, in lichen planus, and certain other forms of lupus allied to the two cases which I have described. I possess no fewer than three drawings, two of my own and one a Hôpital Saint Louis photograph, which show streaks running down the ulnar border of the forearm exactly in the position which we have just seen in our patient. In all these three the morbid process appeared to be of a lupus character. I have other portraits showing ichthyosis running in a line from buttock to ankle, just as we saw the nævoid streak in our patient. These streaks cannot be explained by any known distribution of nerves and what the anatomical condition is which gives them their peculiar position remains, for the present, a mystery. They have been noticed, I know, by other observers as well as myself.

Case illustrating the nature of Syphilitic Lupus.

A stout, pale-faced woman, who had been brought by Mr. G. W. Sequeira, had her nose, upper lip, and adjacent parts dimpled by conspicuous white scars, and had patches of a dusky lupoid inflammation beneath her nostrils and on the upper lip. She said that she had suffered from the disease for five years, and that three times under treatment it had got quite well, but always relapsed in winter. She had a healthy white scar on one forearm and another on one thigh, where similar disease had formerly existed. She had been twice married and had two healthy children, born of her first husband. Two of her sisters had died of consumption. There was no definite history of syphilis, but Mr. Sequeira stated that he had cured the patient of a troublesome laryngitis three years ago with iodide of potassium, and that he had considered the disease syphilitic at the time.

Comments. This case is of much interest in reference to the relationship between syphilitic lupus and lupus vulgaris. Unquestionably I should regard the appearance of the patches and the scars as indicative of syphilis, and Mr. Sequeira's evidence supports this diagnosis. The patient has, however, no history of primary or secondary symptoms, and if she be syphilitic it is probable that the disease was derived from conception. I fully accept the diagnosis of syphilis, but let us note that the lupus has in some features conformed to the type of the tubercular variety. It has been worse in cold weather, and is said to get almost well in summer. There is also a distinct history of tuberculosis in the patient's family. My theory of syphilitic lupus is that there is always a partnership between the tubercle bacillus and the syphilitic virus, and that in all cases in which the diagnosis is difficult it is so because the result is a mixed one. I do not think that we ever see unmodified and well characterized lupus vulgaris in syphilitic subjects. What we see in them is always syphilitic lupus. The well-established principles of treatment are quite consonant with this view; for if you treat syphilitic lupoid affections on much the same principles that you would tubercular lupus, you will as a rule cure them much more quickly than if you prescribed specifics alone. I by no means deny the value of specifics, but I assert that they often fail, as in the case before us, to effect a perfect cure, whereas a liberal cauterization almost always succeeds.

Question of Diagnosis between Lupus and Hereditary Syphilis.

This patient was brought by Miss Webb, M.B.,

on account of the difficulty in diagnosis which it presented.   The patient, a girl of 14, had a large ulceration spreading over her right eyebrow in the horse-shoe form, and with its borders everywhere concealed by a pus crust.   There was a similar and yet larger ulcer on the left forearm.   In each instance a scar had been left where the disease was receding.   The gums of the upper jaw were much swollen, bulging in fleshy masses between the teeth.   Almost the whole of the palate was involved in superficial granular ulceration, and the patient spoke hoarsely from implication of the larynx.   There were scars of former ulcers, multiple, on the legs.   The family history was that the father had died of phthisis, and that the mother had lupus of the face.

Comments.   The condition of the ulcerated patches is certainly much more like that of syphilitic lupus than of the tubercular form ; and I would appeal to their aspect as proof that it is in some cases impossible to diagnose between the two by the appearances presented.   We are obliged to take the history and the concomitant conditions into consideration.   In this case I have no doubt that the disease is tubercular lupus. The patient does not in teeth or physiognomy show any indications of inherited syphilis.   There is a strong family history of tuberculosis, and above all the conditions of the palate and gums are quite characteristic.   It is not very common to find lupus extending to the larynx, and when it does so it is almost always secondary to disease of the palate.

Lupus Necrogenicus.

The subject of this case was a dentist, aged 40, sent by Mr. Hitchins.   He was a delicate-looking man, the subject of a cough, and had on three or four occasions spat blood.   His mother and a sister had died of phthisis.   He had a large patch of lupus necrogenicus on the left index finger, which he attributed to having scratched the finger three years ago with an oyster knife.   The sore caused festered and never healed.   It had been slowly spreading and had been always inflamed in winter, being, when seen, at its worst, in connection with the recent cold weather.

Comments.   The chief interest of this case is as to the origin of the lupus.   Did the man infect his finger, at the time of the injury or afterwards, and introduce from without the tubercle bacilli?   or, on the other hypothesis, were the bacilli latent in

his system and merely took advantage of the local injury to develop ?   The injury was a mechanical one, and not likely to have conveyed any poison, necrogenic or otherwise.   Although the man's occupation as a dentist may have been supposed likely to expose him to contagion, yet we have his assurance that at the time he was not working at it. I may confess that I am not myself inclined to search very far for sources of local contagion in tuberculosis of the skin.   The theory that the bacillus is latent, and that a mechanical injury produces the conditions that permit its development, seems to me to cover the ground far better. Here is a man who is himself probably phthisical, and whose mother and sister have died of phthisis. What more likely than that the tubercle bacillus has existed from birth in his tissues, ready to take advantage of any local degradation of vitality.   I admit, of course, that tubercle may be inoculated. Of this we had a very good example in the tattooing cases which were recently demonstrated here.   At the same time I do not believe that in one in ten of the cases of strumous affections of the skin and mucous membrane (including lupus), there has been any inoculation.   In the great majority of these cases the injury which excites the disease is a mechanical one only.

Case of Malignant Syphilis.

A man, aged 25, introduced by Mr. Sequeira, who appeared to have nearly died during the secondary stage of syphilis.   He was in a condition of extreme emaciation, and only just able to walk.   He was covered with the scars of rupia, and had still some considerable ulcers.   The duration of his disease was only eleven months.   He described the original chancre as having been small and insignificant, and only lasting a few days.   It was for an inguinal bubo that he first consulted a surgeon, and his disease was not recognized as syphilis until the eruption appeared. The eruption was described by his medical attendant as very like small-pox.   It was attended by such illness that the patient was obliged to keep his bed, and had on several successive days a temperature of 103°.   The throat symptoms were severe, and were soon followed by iritis, first in the right and then in the left eye.   On May 1st, his left eye having been destroyed, it was excised at St. Thomas's Hospital.   He was ten weeks in St. Thomas's, and during most of the time was covered by rupia, and suffering from a severely ulcerated

throat. Recently he was supposed to have lost power in his right arm and left leg. On examination of these limbs it appeared probable that the supposed defect of power was due to inflammation of joints.

Comments. I do not know that I have ever seen a case of syphilis which better deserved the epithet " malignant " than this one. We have, in the first instance, an eruption resembling variola, and attended with high temperatures, next rupia and inflammation of the eyes of such severity that one of them was lost. No doubt specifics had been judiciously used, for the man had had excellent advice throughout ; but probably he was one of those in whom specifics do not act favourably. There are a few such. Let me ask you to observe, however, that it is not a case in which specifics are in any way responsible for the severity of the symptoms. They were not used until the eruption was fully out, and the idiosyncrasy of the patient in reference to the syphilitic virus was declared. The question now is, what treatment can save him ? He tells us that whilst in St. Thomas's Hospital he got much better, and that he was almost free from his eruption when he left, but he has relapsed since. I should advise, in his present condition, that he should, if possible, be sent to the seaside, and that mercury in combination with tonics, and above all with opium, should be freely used. I would for the present avoid the iodides ; but it is quite possible that further observation of the case might alter my opinion in this matter. One or other specific he must have, or a fatal event is threatened. Quinine, opium, and sea air will, I feel confident, enable him to bear mercury to an extent which might under other circumstances be prejudicial. Sea air alone will do him no good, but it will enable him to bear the remedy.

Molluscum Contagiosum; Contagion from a Dog.

A young woman, aged 23, was brought by Mr. Hitchins to illustrate the fact that molluscum might occur on dogs, and prove contagious from them to the human skin. In the patient the molluscum spots were present in great numbers over the chest and shoulders. The face, as usual in adults, was free. They had been present about two months. In inquiring as to the source of contagion, Mr. Hitchins had been told that no other cases had been known in the house, and had been asked whether it were possible to ac-

quire the disease from a dog. The patient having at her next visit brought her dog, Mr. Hitchins had ascertained that it did certainly exhibit on various parts of the skin very characteristic molluscum spots. Both the patient and her dog attended for demonstration, and from the latter some of the molluscum spots were excised for examination. They were exactly like those obtained from the human skin, and under the microscope the molluscum bodies, psorosperms, were found in abundance. On the dog the molluscum spots were for the most part isolated. They occurred scattered in small numbers here and there over its back and sides. Some of them were as large as small peas, and they were in various stages of precisely the same processes as are frequently observed on the human skin. Some were becoming pedunculated and likely to drop off, whilst others were inflamed and crusted. No precise date could be assigned to the disease in the dog, but it was supposed to have suffered for two years.

Comments. We are much indebted to Mr. Hitchins for bringing before us such an interesting, and, so far as my knowledge extends, unique demonstration. Further inquiries in this direction must be made, for it may be that our dogs and cats often suffer from this malady and become sources of contagion to their owners. In some instances the transference of the disease may be in the reverse directions. English authorities have long been almost unanimous that molluscum contagiosum is really contagious, and that it spreads by contagion only. For long we did not know what the nature of its contagious element was, but recent researches have made it almost certain that it is of the nature of a psorosperm. These psorosperms not improbably explain also the occurrence . of certain forms of contagious lichen, and thus molluscum contagiosum will probably soon stand at the head of a natural group. It may be that in all these the contagion is sometimes derived from our domesticated animals. A very curious fact as regards molluscum is that it appears to be of but limited duration. In the case of the dog we have just been told, though not with any great certainty, that the spots have been present for two years. This is an unusually long period, and I do not think that I ever, in a single instance, saw molluscum contagiosum on the human skin with a history of such long duration. Why it should be of

limited duration I do not know, but the fact is certain that, whilst we see plenty of cases of a few weeks, or a few months, we do not see any in which it has lasted much longer. If it should ever be in the power of any one present to produce a patient in confutation of this statement, we shall all be much interested in seeing the case.

REVIEWS.

Gould's Large Medical Dictionary. (Blakiston, Son & Co., Philadelphia; Balliere, Tindall, and Cox, London.)

Published at £2 nett.

Books are not often met with which fairly baffle the capacity of a reviewer to adequately estimate and to describe, and in which excellence is the source of the difficulty. Yet this is the case with the work before us. In the literal truth of the words, it is a book in which the reader is "lost in admiration," to. find himself gain, however, in appreciation when using it.

In 1623 double-columned small quarto pages are given a number of words used in medicine, medical science, and sciences connected less closely with medicine. The number is not stated, but there cannot be less than 30,000 words arranged in separate paragraphs, and a total, including the words given subordinately, but in different type, in the course of the paragraphs, of 70,000. The type is very clear, and reference easy.

The pronunciation of every word is given, as well as its derivation, followed by a brief, accurate, and useful description of that which the word signifies. The reader not only has the word defined; but he can also gain a clear, distinct knowledge of what the word denotes. But there is more than this in many cases. On some subjects the complexity of the terms has made it necessary to give what is really a miniature treatise, in order to afford information upon each of the many names employed. Indeed, the many pages devoted to microscopic staining would be objected to as excessive but for the astounding number of various stains which have to be described.

Of great value is another feature which has never been carried to such a degree, or elaborated with such care—the numerous tables showing the various relations, for instance, of muscles and nerves, tests, etc., all carefully compiled and certain to be found of service. The figures of everything that it is necessary or advantageous to illustrate are original, and all are good. Lastly, the general form of the work, the character of the printing and binding, compare, and more than compare, with that to which we are accustomed. Perhaps, however, this impression is increased to a degree which precludes comparison by the convenient arrangement of cut-out thumb spaces in the margin, each bearing the letter to which it opens. The convenience of this system is great.

Of course such a book cannot be produced except at vast expense, but it is quite certain that at a still higher price than that at which it is sold it would be cheap.

Gould's Student's Medical Dictionary. 8th Edition. (Blakiston, Philadelphia; H. K. Lewis, London.)

Published at 12s. 6d. nett.

It is strange that a book of this character, which has reached the 8th edition in America, should be so little known in this country, where, moreover, it has no real rival. But a comparison of this and the larger "Illustrated Dictionary" of the same author, suggests a doubt whether the term "8th edition" is not somewhat formal. The "Student's Dictionary" contains, in about a quarter of the space, all the essential information given in the larger work, without, however, the illustrations, which add so much to the value of the fuller descriptions. Yet the smaller work contains a thousand columns, with an average of 20 terms not only defined but explained in every column, and the student—or for the matter of that the practitioner—who needs to know the meaning of a word which is not contained among the 20,000, is certainly for one reason or another, outside the range of practical consideration. Not only does it contain many of the useful tables of bacilli, arteries, muscles, etc., but at the end are brief summaries of the chief facts of vital statistics. A conception of the general character of the book can be conveyed best by an extracted article, taken quite at random :—

"Cura'rè. Woorara. A vegetable extract obtained from Paulinia C. and certain members of the Strychnos family. A powerful paralyzer of the motor nerves and the voluntary muscles. Used in S. America and elsewhere as an arrow poison. In toxic doses death occurs by paralysis of the organs of respiration. Reported effectual in two cases of hydrophobia, and has been successful in tetanus. Dose by hypodermatic injection gr. $\frac{1}{30}$—$\frac{1}{4}$."

The precision and the brevity, and yet adequacy of the account of that which the word indicates is typical, and may safely be left to utter its recommendation. The book has no rival.

A CLINICAL LECTURE

ON

TWO CASES OF INTRA-THORACIC TUMOUR.

Delivered at St. Bartholomew's Hospital, Feb. 22, 1895,

By SIR DYCE DUCKWORTH, M.D., LL.D.,

Physician to the Hospital.

GENTLEMEN,—I propose to call your attention to-day to the subject of intra-thoracic tumour, and by a strange coincidence we have just now in one ward two cases illustrating this condition. Intra-thoracic tumours are generally difficult of diagnosis. They are obscure, especially in the early stages, and, like all other morbid conditions, their exact nature is to be made out and ascertained by means of the symptoms of which the patient complains, and especially by means of the physical signs which can be elicited by careful examination. From the symptoms and the signs, therefore, we make the diagnosis. In the progress of these cases the diagnosis generally becomes more easy ; the symptoms become more prominent and more urgent, and the physical signs more obvious and readily recognizable.

Let me mention first—to remind you of the nature of the commonest forms of swelling or tumour in the chest—that there are mainly two great divisions : intra-thoracic tumours due to enlargement of blood-vessels, the commonest of which, of course is that condition we know as aneurysm ; and the occurrence of a tumour which is, in reality, a new growth, most often of malignant quality, sarcomatous or carcinomatous and some-times of a lymphomatous nature. To a case of the latter disorder, that is, a mediastinal, or intra-thoracic tumour due to lymphoma, we give the name of Hodgkin's disease—one affecting the glandular structures more particularly. These, then, are the commonest conditions which give rise to intra-thoracic tumours : first, aneurysm, and secondly, new growths, generally malignant in quality, and even if they are lymphomatous, this quality, for the most part, attaches to them.

I think I shall be able to show you to-day two patients which illustrate the two commonest forms

—the first illustrating aneurysm, and the second new growth, probably cancerous in nature.

We have before us a man, Charles Jones, aged 47, who was admitted on the 28th January. He is a dock-labourer by occupation, and accustomed to hard and heavy work. There can be no doubt that all cases which present signs of intra-thoracic tumour require a great amount of careful attention, and a minute study of the details concerning the physical signs in the chest. You cannot make a diagnosis of these cases in a hurry, because you have to examine all parts of the chest carefully. You must take note of the sounds elicited on percussion and of those which are heard on auscultation, and you have to gauge the physical signs which, for the most part, are pressure-signs. All this takes time.

The symptoms in all cases of intra-thoracic tumour may be enumerated as follows : pain ; difficulties of the circulation in the head and chest more particularly ; and difficulty in breathing, or dyspnœa. The physical signs in such cases, due to aneurysm or new growth, are for the most part what are called pressure-signs, indicating an interference with the blood currents, compression of the thoracic organs, the trachea, the bronchi, the lungs, the heart and pericardium, the great veins, the lymph-channels, and certain nerves. The progress of the growth leads to displacements of the organs, bulging and prominences of the thorax, generally great venous fulness, or plethora, and sometimes, particularly in the later stages, effusion of fluid into the pleura,—a condition which you can naturally understand would complicate the diagnosis very much, especially if you were called to see a patient for the first time in a late stage, with both solid tumour and fluid superadded to it. That is a very difficult condition to diagnosticate.

Taking the case of this patient before us, we find that four months ago, while he was sawing a log of wood with a large two-handed saw, he was seized with a sudden gnawing pain in the pit of the stomach, going through to his back, which took his breath away and obliged him to stop work. He has been unable to follow his occupation since on account of the pain which exertion brings on. The pain is always in the same place, and only

comes on with exertion. It is not felt when he is at rest, nor is it influenced by food. When the pain comes on it is always accompanied by short-ness of breath, and since it began this has not increased. About a week after the first attack of pain he first noticed that the veins in front of his chest became large. On admission he said they were much larger than they were when he first noticed them. A month later the veins on his belly began to enlarge, and all these veins have steadily got larger on exertion since the first occur-rence of pain. Distended veins have appeared upon his face, which has become larger, more bloated, and congested; and he finds that his body gene-rally has become thinner. He has had some slight cough, of no special character or quality; it was not particularly noisy, and there was no loss of voice, but he thinks his voice has become weaker than it was. In his past history I find that he drank freely of beer—five or six pints a day—that he worked hard and laboriously, and that fifteen years ago he had syphilis, the scars of which are still detectible. We must also notice in his past history the fact that two years ago he had a fall from a height of some fifteen feet, which rendered him insensible for three months. It left no paralysis, however, and at the end of that time he was able to return to his work. It is necessary to recall that fact, because it may have something to do with his present condition. You can now inspect the patient, and judge of the aspect of his chest. You observe a great enlargement and fulness of the venous system. You notice the enlargement of the external jugular, which has in-creased in size to the diameter of a thumb. You can also see the general fulness of the neck, and the bloated vascular condition of the face. Coming down to the thorax, you may observe enormously dilated and very tortuous veins, twisting down in corkscrew-fashion over the thorax, and joining the superficial abdominal veins, and others, the tortuosity and dilatation diminishing from above downwards. The uppermost veins are the fullest and most tortuous, and the lower ones are less full and less tortuous. He has had varicose veins in the calves of his legs for some years, but there is nothing remarkable in the condition of the veins in his thighs.

Judging from the bloated, full, turgid appear-ance of his head, and from the great localized turgescence of his venous system, you may be per-fectly certain that there must be some obstruction of the venous flow into his chest, that the blood cannot flow along the two innominate veins, and cannot reach the superior vena cava, and therefore the other veins in relation to the chest have had to enlarge to meet the difficulty, and to do their best to carry on the circulation in this way. Thus we have the blood struggling down the superficial integumentary veins over the mammary region to join the superficial epigastric veins, and so to get back into the inferior vena cava—probably through the iliac veins. It is not difficult to tell what direction the blood is taking in these obstructed veins. You might tie a cord round the patient so as to compress the veins, in which case you find an enlargement all above the cord and very little below, or you simply empty one tract of vein with two fingers, pressing the blood out, ascertaining in which direction the blood flows most quickly back again. It is quite easy to see that the blood is hurrying in this case from above downwards. Not being able to get back through the large vessels of the upper part of the chest, it has to come back by new channels which it has made in the course of time. The apex-beat of the heart is not felt, but the percussion dulness indicates that it comes out nearly as far as the nipple-line. Since the patient came in an area of dulness has been found to spread across the chest, passing over the right side of the heart to the line which you notice has been marked on the patient's chest, and which represents the uppermost line of dulness furnished by the liver. It will be quite impossible to go into all the minute details of the physical signs in this case; but it will be easy for you to compre-hend the details from the lines mapped out on his chest. The area included in the circle indicated on his back occupies parts of both interscapular regions, and is dull to percussion. All the other parts of the back representing the lungs are per-fectly clear and resonant. The respiratory note, heard in the parts corresponding to the upper lobes of the lungs and the interscapular regions, has a peculiar tubular or bronchial quality—a note which plainly indicates compression of the lower part of the trachea, so that both bronchi are in-fluenced by it—the right more particularly. The breath-sounds are altogether diminished in inten-sity. The amount of air entering the lung is, no doubt, not as large as it would be in health. You notice these large spots on the face and shoulders, which are the result of the medicine he has taken.

Those are the main features in outline—and

necessarily in outline—of the case. The indications afforded by them are as follows : First, we take note of his sex; next, his age, 47; his occupation, a heavy and laborious one; his previous habit of taking a good deal of beer, and the occurrence of syphilis in his past life : all these factors, I think, help us to form the opinion that this man is suffering from aneurysm—that is, the intra-thoracic tumour is of an aneurysmal nature.

It is impossible, however, to be certain of this. One has to make a good guess, and a good clinical guess is a very good thing. You will often have to guess at your diagnosis; and your guess will always be perfectly justified and honourable, provided it is arrived at by careful examination and consideration of all the phenomena and the history of the case. A guess, however, is not justifiable or trustworthy when it is made in a hurry, and on insufficient data. On the whole—but of course I may be mistaken—I feel inclined to believe that this man is suffering from aneurysm. It is interesting to note that when he came into the hospital I was prevented by illness from attending to my wards, and Dr. West took charge of this patient, and formed independently the opinion that this man's symptoms were due to aneurysm. When I resumed my duties, I was in happy ignorance of Dr. West's diagnosis: it was only after I had formed my own opinion that I learned what Dr. West's views were. It is always satisfactory to learn that a person, qualified to give an unbiassed and independent opinion, has arrived at the same conclusion as oneself. I call your attention to the age of this man more particularly, as it is near what is called the aneurysmal age, which is about 40. More aneurysms occur in the decade between 40 and 50, than at any other time. Most aneurysms occur from 39 to 47. I have often told you in this theatre, that amongst the great destroyers of blood-vessels and arteries are syphilis, alcohol, and strain. Putting these three together (we have them all in this case), we have just the conditions which favour the onset of aneurysm. You remember I told you that this man had a bad fall and was insensible for three months. A man with a degenerated aorta or blood-vessels may quite well, under the stress of a strain and heavy fall, suffer some slight crack in the blood-vessels. Decayed arteries become either tough or brittle; if brittle they are apt to crack and break. And so it may be that the very commencement of this man's trouble may date from his fall. But more specifi-

cally in this case the factor of strain is brought out in the great stress and labour of his work at the double-handed saw. That may quite well have caused a sudden enlargement of a small tumour already in progress. From that time, probably, more active growth has taken place in the aneurysm, and given rise to the symptoms we have noted. I am strongly of the opinion of those who think that syphilis is one of the great predisposing causes of aneurysm. There can be no question that syphilis figures in an enormous percentage of cases of aneurysm in both sexes; in fact, so rare is aneurysm in women that it has been said, and I think with truth, that you may not expect to meet with an aneurysm in a woman unless there has been a history of syphilis. That seems a strong statement to make, but it throws a flood of light on the occurrence of aneurysm in men, aneurysm being exceedingly infrequent in women and almost entirely a man's disease—men being the victims of the worst forms of syphilis in greater numbers than women.

All these points, I think, tend to illustrate the nature of this patient's intra-thoracic tumour. Nothing is more difficult sometimes than to make a diagnosis of thoracic aneurysm. It is easy to make such a diagnosis when there is a bulging tumour with impulse and loud bruit; but when an aneurysmal tumour is deeply seated in the thorax, far away from the reach of all touch, and gives rise to no morbid sounds, then the diagnosis becomes exceedingly difficult. Aneurysms may go on for months, and sometimes for years, without being recognized, although they may be suspected by reason of pain, dyspnœa, and increasing pressure-signs.

There is something more in this case which I have not yet mentioned. Dr. Garrett, my house-physician, discovered, on a careful examination of this man's larynx, that the left vocal cord was paralysed. Paralysis of the left vocal cord means some interference with the left recurrent laryngeal nerve, which curves round the arch of the aorta. It is quite clear that that nerve has become involved in this intra-thoracic tumour. One of the commonest causes of such a palsy is an aneurysmal tumour. Aneurysms in the thorax very often cause pressure on the sympathetic nerve, and, therefore, changes in the size of one of the pupils; but this patient has had no inequality of the pupils, so we have no reason to suspect that there is pressure on the sympathetic. On

examination there is found an inequality of the pulses, the left being smaller, the blood current through the left subclavian being interfered with, and the pulse being a little delayed and smaller than on the right side. There are no signs of pressure on the œsophagus, and therefore no difficulty is experienced in swallowing. I think, probably, that this man has a large sacculated or pouched aneurysm, coming off from the hinder aspect of the transverse arch of the aorta, and there compressing the lower part of the trachea and bronchi, and being also in a position, and large enough, to compress the superior vena cava, and to interfere with the left recurrent laryngeal nerve at the point at which it turns round the vessel; and this condition, I think, gives rise to all the symptoms which we have in this patient. That, then, is the diagnosis of this case. The heart is not displaced, or hardly so. The effect of even a large aneurysm is *nil* upon the left ventricle. Many of you might expect to find hypertrophy or dilatation of the left ventricle behind a large aneurysm. Nothing of the kind happens. There are no cardiac murmurs, and no signs of valvular disease. One sign of an aneurysm is missing in this man—a most marked and useful sign—viz., accentuation of the aortic second sound.

In most cases of thoracic aneurysm the second sound over the aorta is exceedingly loud, clanging, and noisy. In the case of this patient, up to a few days ago this second sound was singularly quiet; but on examining him yesterday it appeared to us that the second sound was beginning to be a little louder than formerly, and to assume a clang-ing quality.

This is our diagnosis, and the treatment, of course, is the treatment for thoracic aneurysm. That is a subject I have often discussed with you, and shall not delay to consider it at length now. It is by restricted diet—restricted solids, and espe-cially restricted liquids—and by the use of the best drug I know of for the purpose, iodide of potassium, in large doses of 15, 20, or 30 grains three times a day. This patient is now taking that medicine, and his diet is restricted both as to solids and liquids. The rash we have already ob-served upon him is produced by the iodide of potassium, and we are giving a little arsenic to lessen this trouble.

The second case now before you is that of a man, aged 46, one year younger than the last patient, a bookbinder's porter, accustomed to heavy lifting work. He was admitted on the first day of this month. His story was that he began to suffer pain and swelling in his right foot and ankle three weeks before last Christmas. The next thing he noticed was swelling in his face, which became full and bloated. Since admission his face has become more red and turgid, and his eyeballs prominent. The swelling in the hands, head, and face has, on the whole, increased. It is generally found worse the first thing in the morn-ing. He has had a slight dry cough for years, but this has not increased. There is no difficulty in swallowing. He has been subject to headache for six or seven years past, but one of the worst symptoms since he began to complain has been a severe headache, very constant, and disturbing to him at night. Three years ago he had a small tumour, the size of a large nut, removed from his forehead, the nature of which we have now no means of ascertaining. His family history does not throw any particular light on his symptoms.

To any one passing by this patient it would be easy to see there was some great interference with the return of blood from his head. You will notice in this patient—though not to the same degree as in the last case—the fulness and tor-tuosity of veins in the thorax, the blood coursing from above downwards, in the same way as in the last case, to inosculate with the superficial ab-dominal ones. This patient is also the subject of a rash upon his skin, the result not of iodide, but of bromide of potassium.

An examination of his chest shows irregular areas of dulness. Since he came in we have noticed a bulging or prominence in the right mam-mary region, presenting very much the appearance found in a person who has long had *morbus cordis*, with great hypertrophy of the heart, the chest-walls being pushed before it. This has come on during the last month. There is an area of dulness covering the mediastinal and sternal region, the heart being somewhat pushed down, and the liver, possibly, also pushed down to some extent, gradually increasing since admission. An area of dulness to percussion, with tubular quality of breath-sounds, occupies the right inter-scapular region, indicating compression of the bronchial tubes and of the right lung. In front these characteristics are not so easily made out. But putting together the irregular patches of dulness, the absence of all signs of interference with his heart and circulatory system, I formed the opinion that this case is one

of intra-thoracic tumour due to some form of new growth. The left pulse is rather smaller than the right one, although this was not noticed when the patient was admitted. We have not observed any enlarged glands, but they may appear later on.

Obviously, this tumour is of a character to impede the return of venous blood from the head, and so we find this man to be greatly oppressed with venous plethora in his head, his face is bloated and purple, and he has very severe headache.

It is of the utmost importance in a case of this kind, to make a careful examination of every superficial inch of the chest, in order to find signs of what is going on. In this case the symptoms indicate great venous difficulties. Stupor and headache are the signs of retarded venous flow from the head. In addition, we have the bulging of the front of the chest, the dull areas, signs of pressure on the bronchi, and on the right lung more particularly. Above those dull areas we have sub-tympanitic areas, which are quite as indicative of consolidation below as the dulness over the patch itself.

There is no help to be obtained from the temperature in either of these cases, there are no febrile symptoms in either. That fact is of interest in a case like this, because we may meet with occasional febrile movements both in sarcoma and in lymphoma. In this instance we did not suspect lymphoma ; perhaps this new growth may be of a carcinomatous nature. In both these patients the urine has been quite natural.

The head-symptoms have been so marked in this second patient that we have considered the possibility of some new growth forming, possibly, within the skull, but we have no clear evidence of that. There is no ophthalmoscopic evidence in the shape of optic neuritis. This man is becoming a little deaf, but that may be due to the venous plethora.

The diagnosis here, then, is some new growth arising at the root of the right lung, either in the bronchial lymphatic glands or in the connective tissue of the mediastinum. If that diagnosis is right, of course the case will go on from bad to worse; the tumour will grow, pressure-signs will increase, and all the other difficulties of the case will likewise increase.

The treatment is to relieve symptoms. We cannot touch the disease. Great relief has been found from leeching the temples occasionally. By applying a few leeches to each temple, relief is given to the venous pressure. That may be repeated from time to time. Bromide of potassium has been singularly useful to this man, and has relieved him in a most remarkable way. He obtained rest at night with it ; it relieved his headache ; and in fact, after a few days, his symptoms were so much improved that we had a suspicion that we were wrong in our diagnosis, and so hoped that the man was going to get well. The bloated appearance of his face was reduced, and his face looked brighter and much better. When we suspended the bromide these symptoms returned, and I am afraid we are not getting all the advantages now that we did at first from it ; but still it has been very useful. The quantities he has taken (gr.xv thrice a day) have induced this bromide rash about his face and shoulders. Phenacetin did not do his headache much good. When he came in he had some mercury and iodide of potassium, which it is always as well to try in obscure cases of tumour, lest by any possibility they should be of syphilitic nature ; but if no good follows in a few weeks you need not go on with these drugs. We are now giving quinine and iodide of potassium together.

In course of time, it may be, some effusion may take place into the pleura, and then the diagnosis would become much more difficult if we were seeing the case for the first time. The fluid thrown out in such a case will probably be more or less blood-stained, but not necessarily. Sometimes new growths of a cancerous nature are accompanied by perfectly clear serum in the pleura.

The termination generally takes place in the first year from the onset of symptoms. One could not wish any such patient's life to be much prolonged. As a matter of fact, it generally proves fatal in the course of a few months.

Time will try the accuracy of our diagnosis in both these cases. They are both as bad as they can be, and the outlook is very sad for both of them.

I have with me an old edition of that splendid classic, Sir Thomas Watson's "Principles and Practice of Physic," of which, I have no doubt, there is a copy in the library. The modern edition does not contain the engravings which I am about to show you. These show very well the appearance presented by obstruction due to intra-thoracic tumour in one case, and by an abdominal hydatid tumour in the other — obstructions respectively to the vena cava superior and to the vena cava inferior. Supposing an obstruction to the vena

cava inferior takes place, the veins begin to enlarge from below, the largest and most tortuous from the lower part of the belly coming up to inosculate with the thoracic veins, which are comparatively small. If the tortuosity is above, the uppermost veins are the largest and most tortuous. It is a curious thing to note that the tortuosity occurs where the current is reversed; it is not natural for the blood to flow down these veins, and therefore they become much more corkscrew-like and tortuous. These cases are very unsatisfactory, but an accurate diagnosis of them is of the first importance. We may not often have an opportunity of seeing such well-marked examples together again, and therefore I cannot but think that they will make an impression upon you. It is a great thing to get a mental picture of cases of this kind, and having got this, it will come to help you when you meet with subsequent ones. Meanwhile, we are doing our best to relieve the symptoms, but not with any hope of ultimately overcoming the disease in either case.

A CLINICAL LECTURE

ON

CANCER OF THE RECTUM.

Delivered at the London Hospital, November 29, 1894,

By FREDERICK TREVES, F.R.C.S.,

Surgeon to the Hospital.

GENTLEMEN,—We are going to consider the subject of a common disease, viz.: cancer of the rectum; and I will say this to begin with, that there is only one form of primary cancer met with in the rectum. It is a form that goes by two names, and those names are interesting because they express some of the chief facts about it. One name is "cylindrical epithelioma," and the other is "malignant infiltrating adenoma." The reasons that led surgeons to give those names are potent reasons, as you will see directly. The term epithelioma is based upon this, that in the first place, the growth concerns the surface, and is comparable to cancer on any other mucous surface, like the mouth. The second reason is this, that when this said cancer of the rectum reaches the anus and becomes visible, it takes the un-

doubted characters of epithelioma elsewhere. Thirdly, the whole progress of the disease is comparable to the progress of accepted epithelioma; and when one finds that it is made up of cylindrical cells mainly, the very term cylindrical epithelioma expresses a good deal that is to be said about the disease.

Then it is called "malignant infiltrating adenoma." What are the reasons for that? It seems a confusion of terms. The reasons are these: The trouble always begins in the glands of the mucous membrane, and, as you know, an adenoma is no other than a tumour developed in glandular tissue. Secondly, in its structure it is mostly like an adenoma; in fact, it is impossible sometimes under the microscope to tell it from a simple glandular growth. Thirdly, undoubted simple adenoma may become cancer. So then these two names carry in themselves some of the chief facts about this form of cancer. When you come to examine the growth microscopically, you find gland-like recesses filled with cylindrical epithelium; you find a crude, rough mimicry of mucous gland tissue, a sadly exaggerated attempt to imitate the structure of those glands, with all that boisterous, flimsy exaggeration which is a feature of cancer.

With regard to other terms applied to this little-varying growth, "scirrhous cancer" is simply a hard cancer, and it is quite true that there are some cases of cancer of the rectum which are sufficiently hard to merit the word scirrhous, but the term must be used in no other sense. If it convey the impression that this form of cancer is identical with other cancers elsewhere, which are called scirrhous, then the term is bad.

By the term "encephaloid cancer," it is assumed that you mean a cancer as soft as brain. Well, there are cases of cancer of the rectum that, although, perhaps, not quite so soft as brain, are yet soft, and to such cases there can be no reasonable objection to employ the term "encephaloid," so long as it is not confused with what was at one time regarded as a special form of cancer, but is now known as round-celled sarcoma.

There is only one form of cancer met with in the bowel, and that is the one I have mentioned.

This cancer of the rectum takes two forms, as you see in practice—the tuberous or rapid form, and the laminar or chronic form. Now, in the tuberous form you find one or more tubers or nodules projecting into the bowel. These are not

necessarily hard; some are quite soft. The softer they are the more rapid they are, and it is a curious thing how these rapid growths of the intestine tend to project into the lumen of the bowel. These masses very soon ulcerate, and when they have ulcerated and broken down they leave crater-like recesses, into which the finger drops when the bowel is examined. It is a form of cancer which has little disposition to contract, and it very soon extends beyond the limit of the bowel wall. It may cause an obstruction by its mere bulk. It is, as I said, rapid, and it is a form of cancer which is met with usually in the younger class of patients.

The laminar or annular form differs in nothing except degree from the form just mentioned; one is rapid and the other is slow. The growth invades the submucous tissue—it spreads in that tissue; in fact, it spreads beyond the submucous tissue, and invades the muscular coat. Like other forms of cancer it follows the vessels. The vessels are disposed transversely, and therefore it travels transversely, tending to form an annulus or ring round the bowel; hence the term "annular." As a mass it remains annular and flat. It contracts, and this contraction is an essential feature of it; it contracts for exactly the same reason that acinous cancer of the breast contracts. Many cells are produced; these undergo fatty degeneration. The fibrous stroma which supports them shrinks—must shrink, in fact, when you remove the cells from its midst—and that very shrinking produces the contraction which is so marked a feature of this form of malignant disease. You can understand that it can only occur in a comparatively slow-progressing growth. The breaking down of the epithelial cells from fatty degeneration, and the removal of the *débris*, must be a matter of time; so there is little or no contraction in the tuberous form. In the annular variety, the growth may appear as a definite thick ring round the bowel, just like a big wedding ring. In other cases the growth spreads up and down the bowel vertically, so that the affected part of the rectum is converted into a rigid pipe or tube, and this variety is not quite so slow in progression or chronic as is the really annular form.

Cancer of the rectum, like carcinoma elsewhere, tends to invade the adjacent parts, to attack the glands, and to produce increasing debility and emaciation.

The clinical characters of this trouble we can review but very briefly. Cancer of the rectum is common, but of course cancer itself is common. The actual frequency of cancer in the rectum is much less than you would imagine. If you take all cases of cancer you will find that only 3.5 per cent. occur in the rectum. It is a little more common in males than in females; and is an affection of middle life and advanced age. There are a few cases recorded in comparatively young people.

One cannot help being struck with the fact that inheritance seems to play a part in this form of malignant disease. In not a few instances the patient will tell you that his father or grandfather, or mother or grandmother died from cancer in the rectum. I have actually had under my care three members of one family, all of whom had cancer of the rectum.

Another point one must notice is this—syphilitic troubles are common in the rectum, but so far as my experience goes, they would appear to seldom lead to cancer. That is the opposite of what you meet with in the mouth and tongue.

I need not tell you that the malignancy of cancer of the rectum varies immensely. The course and the symptoms also vary greatly.

What is the average duration of life? Two years; that is to say, taking cases that have not been treated by any radical measure. What is the possible limit? The possible limit, without radical treatment, may be put down as between four and five years. Death will be due usually to exhaustion, obstruction of the bowel, or peritonitis. These are, I believe, in order of importance, the commoner causes of death.

The onset of cancer of the rectum may be very insidious—so insidious that patients, when they have come to a medical man for the first time complaining of the rectum, may complain of very little, yet when the surgeon examines the bowel he may be perfectly amazed to find the progress the growth has made. The earliest features of cancer of the rectum—those that you have to look for—are, speaking very generally, these :—Uneasiness of the bowels; a sense as of a foreign body in the rectum; and a sense of imperfect relief when the bowels have acted. Straining, too, is another early symptom; and it may be for a while the only one. A pain in the back is often another early symptom. Blood in the motions has now and then been the first thing that the patient has noticed. One of the most important of the early

symptoms I should especially mention, is an unaccountable diarrhœa. It is needless to say that the rectum should always be examined in all doubtful cases of diarrhœa in middle-aged or aged persons.

The fact that the rectum is in relation with the sacral plexus will enable you to understand that, now and then, the first symptom of cancer of the rectum has been sciatica.

With regard to the symptoms in detail :—The pain in cancer of the rectum varies, and at first it is usually quite slight. It is perhaps most severe when the anus becomes involved. It is also very distressing when it extends to the parts around the sacral plexus, or to the bladder. In such cases, there may be intense neuralgia, cramps in the limbs, severe sciatica, or intolerable bladder troubles, or troubles and pains due to an opening into the vagina. Later on, may come the pains which attend obstruction of the bowels. Bleeding is common ; but it is seldom marked. There are few patients with this disease who have not bled from the bowel, but there are very few who bleed copiously. As a matter of fact, the cases are generally marked by what would be called a foul, bloody discharge.

With regard to the condition of the bowels, that which is more usually met with is that of constipation, alternating with spurious diarrhœa. Spurious diarrhœa means this:—There is an obstruction in the bowel which is not complete ; fæcal matter lodges above that obstruction ; it decomposes; it irritates the bowel; a copious catarrh is the result of that irritation, and this catarrhal fluid escaping through the stricture, bringing with it dissolved fæcal matter, produces the symptom known as spurious diarrhœa. In due course, the patient loses control over the sphincter, the anus becomes patulous, and there is then an almost constant discharge from the bowel. In nearly every case there is a discharge of pus which can sometimes be recognised as such ; at other times it is lost in the bloody discharge.

With regard to obstruction of the bowels, I must first say that it is not inevitable. Many patients go through the whole course of cancer of the rectum, and die of it, without having had obstruction of the bowels. When obstruction does occur, it is of a chronic type as a rule, with all the symptoms that mark obstruction of the colon. Now and again the symptoms are acute, but such cases depend upon the somewhat sudden blocking of the narrowed part of the colon.

When you examine the patient with the finger, you encounter in the rectum a narrowed strait that is bounded by a hard, irregular, nodular surface. The condition may be more marked on one side than on the other. In some cases you can make out a definite stricture—a definite narrowed passage, which has been compared to the os uteri. The bowel around is hard, and if the disease be at all advanced the bowel is fixed. The edge of the growth is raised, hard, and everted. If there be any difficulty in reaching the growth in the ordinary recumbent position, the patient should be examined standing up.

I would say nothing about the differential diagnosis except to point out that other forms of ulceration of the rectum may be mistaken for cancer. One should . notice this difference—in syphilitic ulceration (to take the commonest form) there is less contraction, less induration, a less marked nodular growth, the extent of the disease is often greater ; and last of all, and most important of all, the bowel remains perfectly mobile.

Now comes the question of treatment. I will speak of three elements of treatment in this order —excision of the rectum, colotomy, and palliative measures.

(1) *Excision of a part of the rectum.* The mortality of this operation, as given by Ball, is 16 per cent.; as given by Kelsey, on the basis of 140 cases, it is 19 per cent. As most successful cases are recorded, and most unsuccessful cases are allowed to pass into oblivion, the mortality is probably higher than the figures given. In the great majority of the fatal cases death has been due to peritonitis.

Now what are the prospects of relief or cure to the patient from the operation. Out of 100 cases of excision of the rectum examined by Mr. Butlin, there were only 13 in which the patients were sound at least two years after the operation. That is a terribly small proportion. Here you have 100 cases of excision of a part of the rectum, a radical operation ; two years go by, and 13 alone out of that number are alive and sound. Mr. Cripps gives 30 cases, with only two deaths. In 10 out of the 30 the disease came back within twelve months, and only 5 out of the 30 were free from disease two years after the operation. You must bear this in mind, that although the operation may not be spoken of as a very dangerous one, it does undoubtedly involve a great deal of after inconvenience to the patient. It is not like an

excision of the tongue, or of part of the lip, which lead to comparatively minor inconvenience. Excision of the lower part of the rectum for cancer very often leads to considerable distress after the operation is done, mainly by reason of incontinence, or by undue contraction of the cicatrix, or by fistulæ.

What are the cases most suitable for excision? They are these: the disease must be recent; it must be close to the anus; the rectum must be movable, and the tissues around the rectum quite free, and it must be possible to remove the whole growth without any question of doubt. What does this mean? It means that the cases of cancer of the rectum suitable for excision are exceedingly few. An estimate has been made that out of 100 patients with this affection who present themselves, only 10 are in any way fitted for this operation. Just look then at the prospects. Only 10 out of 100 of the victims are suited for the operation, and when the operation is done 16 to 19 per cent. die; in a great majority the trouble comes back within a year, and in just a very few the cancer may be said to be brought in abeyance. Kelsey collected 100 examples of excision of the rectum, and out of these there were only 6 cases in which it could, I think, be honestly said that the patients were cured, and there were 24 who were sound at a period varying from one to six years after the operation.

(2) *Colotomy*. The ordinary operation done for cancer in the rectum is that known as inguinal colotomy. It is an operation attended with comparatively small risk to life. The risk may be put down at, we will say, 3 to 4 per cent. Now, the real problem connected with this measure is this: Should colotomy be done at once—that is to say, as early as possible—or should it be carried out at a later period, or, in other words, postponed as long as possible? This is a question that constantly crops up in practice. The arguments in favour of early colotomy are mainly these four: First of all, it is said that the operation retards the growth of the cancer. In this connection it is urged—first, that as the rectum is no longer employed, it is not disturbed by the passage of motions; the growth, therefore, is not disturbed, and its progress is thereby retarded. Secondly, by performing the operation, the growth is no longer irritated by the actual contact of fæcal matter; thirdly, there is no pain due to the act of defæcation; and fourthly, obstruction of the bowels is

rendered impossible. These are the four main arguments in favour of an early operation.

Now, what are the reasons against it—or, rather, how are these four points to be answered? With regard to the first contention, I think that there is absolutely no evidence whatever to prove that colotomy will modify the rate of growth of cancer in the rectum either in one way or in another. As to the second point, fæcal matter, we will acknowledge, is an irritating substance; but those who use this argument in favour of early colotomy seem to forget that fæcal matter is the proper material to find in the rectum. The rectum is acclimatised to it. Here is an ulcer in its own home, and surrounded by no other "atmosphere" than that normal to the locality.

This argument, from a commonsense point of view, cannot be allowed to pass. Then, thirdly, it is claimed that after the operation there is no pain from defæcation. Bear this in mind, the bowel between the artificial opening and the anus does not close and dry up; a certain amount of fæcal matter passes by the aperture in the groin, and it is a great mistake to imagine that after colotomy the patient ceases to recognize the existence of his rectum. The bowel above the growth still produces mucus, and often in considerable quantity. That has to find a vent, and defæcation is not brought absolutely to an end. Then, fourthly, the argument is urged that obstruction is warded off by the early operation. But remember this, that obstruction, as a rule, comes on with due notice; the symptoms develop slowly. It is not so acute but that one has plenty of time to deal with it. This item in the argument may mean this: Let us do an operation for the purpose of warding off an accident which may never happen.

What are the arguments in opposition to early colotomy? They are these: Colotomy is only of service in obstruction of the bowels and in pain due to defæcation. If a patient have obstruction of the bowels or pain due to defæcation, colotomy is undoubtedly the right measure to adopt; so that the position in regard to late colotomy is this: Wait until the patient has obstructive symptoms or definite trouble due to defæcation. Another argument against early operation is as follows: The procedure often involves a considerable amount of after distress to the patient; that cannot be denied. You cannot pick out isolated cases and say, "Here is a man who has had an early colotomy performed, and who has been doing his work for

years with perfect comfort." There are such cases, but they do not represent a standard. There *is* a great deal of discomfort after colotomy. Sometimes the distress after this operation is lamentable. I quite acknowledge that in other cases it is comparatively slight, but in one way or another the distress after the operation is a factor which must be considered.

The most weighty argument against the operation is this: No colotomy will relieve those most distressing pains of cancer of the rectum which depend upon implication of the bladder or sacral plexus; this distress, which exceeds any other, is untouched by colotomy.

Acting on the basis of my own experience, I would advise you to discourage early colotomy for cancer of the rectum, and I feel confident that the judgment of the surgery of the future will be decidedly against this measure as an early and routine method of treatment. It would be wearisome to attempt any details of all the cases which have led up to this conclusion, but I want to mention two at least which will illustrate the point.

One case was that of a man of 57. His cancer was first discovered in March, 1891. In August, 1891, the patient was very strongly urged to have colotomy performed. He was told that he could not live unless the operation was at once done, and that what little remained of his life would be lamentable. I saw the man for the first time in September, 1891. The stricture then readily admitted my forefinger, and the patient weighed 12 stones. I strongly advised him to depend for the present upon palliative measures. He was the manager of an important public company, and he had so far never lost one single day of attendance to his duties. He acted upon my advice. He continued his work in the south of England, coming to London frequently on business. He did all his work as well as ever for the rest of 1891, for the whole of 1892, and indeed up till June, 1893. It was not until that date that I did colotomy. I have no hesitation in saying that the patient was infinitely better during the long period than he would have been with a colotomy wound. Until a few days before the operation he was actively engaged in a very exacting work, and the operation was only carried out when symptoms of obstruction appeared. As soon as colotomy was performed he gave up his appointment, and died nine months after it was done.

As a matter of fact, the cancer did not grow at the fearful rate prognosed by those who advised early colotomy, and the patient was undoubtedly a gainer by not having the operation performed until it became necessary.

In another case, the patient was a clerk in the Post Office, and 56 years of age. His cancer was discovered late in the year 1892; in January, 1893, he was most strongly urged to have colotomy done on the ground that the growth would rapidly increase, his life—without the operation—would be shortened, and all sorts of distressing consequences would follow. I saw him in May, 1893, and advised him to adopt palliative measures, and to leave colotomy until it was required. I have seen the man recently, and cannot avoid asking, what would have been the result if he had had colotomy done? He was a clerk in the Post Office, and he could scarcely have gone to his work again, what with the persistent dressings, the possible soreness of the skin, the offensive smell, frequent discharge of fæces, and one thing and another. What is his condition now? He declares to me that he has not lost weight, and that he has not lost one single hour's work. He has attended his duties regularly, and the amount of inconvenience his rectum gives him is comparatively trifling, and yet colotomy was strongly urged more than two years ago; the growth has not spread at a great rate, nor has it caused obstruction, as the patient was told was inevitable.

Those are two cases out of a number I might quote, in which these prophecies have not been fulfilled.

The palliative treatment I would urge is this:— First, the daily passage of a soft olivary bougie. It is useless to add that this must be done with care. It can hardly be done when the cancer is beyond the reach of the fingers. In the two cases I have mentioned bougies were passed every day, perhaps missing a day occasionally. Did it give them infinite distress? Not in the least. It just kept the canal patent for a time.

The next thing is this: Many of these patients have defective teeth. The food is not masticated, lumps of undigested matter pass into the bowel, and, of course, obstruction follows. These patients must never have any gross *débris* in the bowel. There must never be any question of anything passing through it that will be much more than a mere fluid. Consequently in these cases especial attention is paid to these points—food, teeth, absolutely regular meals, and the most careful selection

of such articles of diet as will leave the least *débris* in the bowel. It is well to avoid milk. Milk will produce in adults the largest scybalæ which can be produced. Into the actual details of the dietary it is unnecessary to enter.

In addition to these measures artificial digestives may be used, such as pepsine; salol, in doses of ten grains, may be administered to lessen the tendency to decomposition within the bowel. The value of the drug in this connection appears to be undoubted. Frequent enemata should be given in the hope of washing away matters collected above the stricture, and the bowels must be kept loose with laxatives. These need to be frequently changed. Cocaine tends to relieve the pain up to a certain time. Beyond that morphia is necessary; but the longer the use of morphia can be postponed the better.

A CLINICAL LECTURE

ON

EMPYEMA OF THE ANTRUM OF HIGHMORE.

By J. ERNEST LANE, F.R.C.S.,

Surgeon to Out-Patients, and Lecturer on Anatomy,
St. Mary's Hospital.

GENTLEMEN,—You have recently had the opportunity of observing in the wards of this hospital several cases of empyema of the maxillary antrum which have been cured by a very simple operative procedure, and I propose to call your attention to-day to the clinical aspect of cases of this nature. You may possibly have observed that this condition is far more frequently met with now than was formerly the case, but it does not necessarily follow that more patients are suffering from this affection than in years gone by, but rather we should ascribe this circumstance to the fact that greater attention has recently been paid to the surgery of the nasal cavities, and consequently cases which formerly escaped recognition are now correctly diagnosed and submitted to treatment.

It is generally admitted that empyema of the antrum may be caused by morbid conditions propagated by and spreading from either the teeth or the nasal cavity, but there is considerable difference of opinion as to which of these sources is mainly responsible for the disease under notice: there is no doubt that caries and kindred affections of the roots of the teeth are potent factors in the causation of the disease, and in one of the cases I recently operated on, this was undoubtedly the cause. On the other hand it can easily be understood that any affection of the mucous membrane of the nasal cavity which gives rise to obstruction of the aperture from the antrum into the middle meatus, must cause retention of the secretion of that cavity with its pathological sequelæ: tension, inflammation and suppuration.

I purposely refrain from dealing at any length with the etiology of the disease, which is, at the present moment at any rate, a matter of theory, but shall proceed forthwith to the consideration of the symptoms and treatment, this being the more practical side of the question, and consequently more appropriate material for a clinical lecture. The symptoms of this condition are somewhat indefinite, and the one, which commonly induces the patient first to seek advice is an offensive discharge from the nose, escaping sometimes from the nostril, at others down the throat, according to the position of the head. An important point is that the smell of the discharge is always painfully perceptible to the patient, an indication that the olfactory region of the nose is unaffected; this feature will serve to distinguish this disease from the majority of cases of ozæna, for, in the latter, the olfactory portion of the nasal mucous membrane being involved, the patient himself is ignorant of his unenviable condition, and possibly wonders why he is kept at such a respectful distance by his friends and relations. Considerable stress has been laid on the fact that the discharge is unilateral, and undoubtedly in the majority of cases it is so; still, in my limited experience of the disease, I have already met with two cases in which both antra were involved.

On making an examination of the interior of the nose, there is one symptom present in the majority of cases: that is, the welling out of pus from the antrum into the middle meatus; this symptom will naturally be influenced by the posture of the patient, and in order to produce it he must be instructed to bend the head forwards and downwards, so as to place the aperture at the most dependent part of the antrum. Obstruction of this opening will of course modify this symptom, and it is then that trans-illumination of the antrum may with advantage be resorted to. To affect this, the

patient is placed in a dark room, and a small electric light is introduced into his mouth, which is then closed; on establishing the current it is found that the facial bones forming the boundaries of the healthy antrum become perfectly translucent up to the floor of the orbit, but if the antrum contains pus, or is lined by a dense layer of granulation tissue, no light can be transmitted through it. I cannot say that I have had much experience of this procedure; but in a patient recently sent to me by Mr. Morton Smale, the diagnosis was arrived at mainly by this means. The absence of translucency of the antral cavity is held by competent observers to be almost infallible as a diagnostic feature of the disease, and it would seem to be quite as reliable as the time-honoured process of trans-illumination for the diagnosis of hydrocele.

Pain is a symptom on which much reliance cannot be placed, unless with the co-existence of other signs of the disease. It may be situated over the antrum itself, may be confined to the distribution of the infra-orbital nerve, or may take the form of severe frontal headache, so that the affection may be referred to the frontal rather than to the maxillary sinus. Often it is not present.

If the antrum is much distended, there may be noticed some swelling of the face, but this is rarely met with; in one of my cases there was considerable œdema of the lower eyelid, possibly from obstruction of the infra-orbital vein.

In some cases the subjects of this disease are anæmic, and complain of failing health and strength, which symptoms may be attributed to the swallowing of the fœtid discharge, and possibly to the long-continued headache.

In order to clear up cases where difficulty is experienced in arriving at a diagnosis, puncture of the antrum may be resorted to, but I have never yet had occasion to adopt this plan.

Many of these cases may be cured by treatment applied to the nasal cavities, but it is not with such I wish to deal in this lecture; they will seldom be seen in the wards, and must be studied in the special department devoted to the treatment of these diseases; the cases which you have recently seen under my treatment are chronic and inveterate ones, in which nothing short of operative procedure will produce any lasting benefit.

All the surgical measures which have been hitherto adopted have for their object the opening and draining of the antral cavity, and this has been effected by one of the following methods:

(1) through the alveolar process after the extraction of a tooth; (2) through the natural opening into the middle meatus, or by puncture through the inferior meatus; and (3) through the outer wall of the antrum, by an opening made above the alveolar process.

The principal objection to the first method is that the seat of disease is approached through the densest part of the bone, where there is consequently great difficulty in maintaining the artificial opening, and where much pain is caused by the endeavours to maintain its patency.

The objection to the second method is that by it the cavity is not opened at its most dependent part, and that the necessary drainage and washing out can only be effected with difficulty.

The third method, which may be described as Heath's operation, is the one I have hitherto employed, in most instances successfully, and was suggested to me as likely to be most thorough and satisfactory by my colleague Dr. Wm. Hill, who has kindly transferred to me for operative treatment a considerable number of these cases from his special department. The technique of the operation is as follows: the patient's shoulders being elevated, and the upper lip being retracted by an assistant, a horizontal incision about an inch in length is made through the mucous membrane at the level of its reflection from the cheek on to the maxilla, and above the first bicuspid tooth; the mucous membrane is then separated from the subjacent bone to an extent sufficient to allow of the crown of a small trephine to be applied; the trephine is then directed upwards, backwards, and inwards, and by its means a small circle of bone, equal in size to the circumference of the little finger, is removed; some surgeons employ a chisel and cutting bone forceps, but the trephine is, in my opinion, preferable. The walls of the cavity are then inspected, and digitally explored, and thoroughly scraped with a Volckmann's spoon, and the *débris* washed out from the cavity by irrigation with 1 ad 40 carbolic lotion; in order to secure drainage of the cavity into the nose as well as into the mouth, dressing forceps or a trocar are thrust through its inner wall, either into the middle or inferior meatus, and finally, as a first dressing, strips of iodoform gauze are introduced. This is removed and renewed daily for the first three or four days, after which time the cavity is washed out daily with a weak antiseptic lotion, a gum elastic catheter

attached to an irrigator or to a syringe being the best means of effecting this; care must be taken to maintain the patency of the opening for at least a month, at the expiration of which time the discharge will probably have disappeared. This slight operation is occasionally attended with severe hæmorrhage, but this usually ceases spontaneously, or can be checked by hot water application and by plugging.

This plan of treatment is adopted by one of the most eminent authorities on diseases of the jaws, Mr. Christopher Heath, and so far it has proved universally successful in my hands; it is free from the disadvantages attending the other methods, for the seat of the disease is hereby rendered easily accessible to local applications, the opening is made through a very thin lamina of bone, and being situated at the most dependent part of the cavity, it establishes the most efficient form of drainage.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Friday, March 1, 1895.

Gumma in the Tongue after Inherited Syphilis. Remarks on the rarity of true tertiary symptoms under such conditions.

Two young women, sisters, aged respectively 35 and 37, attended so as to demonstrate the absence of peculiarities in physiognomy, etc., in inherited syphilis. In neither was there anything in the least recognizable. In both, the bridge of the nose was narrow, and the features well formed. One of them, the younger, had her central incisor teeth typically misshapen, but to such a slight extent that they might easily have escaped notice, whilst in the other the teeth showed no peculiarity. In neither of them were the teeth in the least indicative of infantile stomatitis from mercury. Both of them were in excellent health, and, with the exception that the eldest had recently developed a gumma in the substance of her tongue, neither of them showed any affection due to their inherited

taint. The gumma alluded to consisted of a lump about as big as a marble, deeply placed in the middle of the right half of the tongue, and bulging both on the upper and under surface. It was tolerably well defined, and not in the least inflamed or tender.

Comments. I have brought these patients forward because it happens to be in my power to speak, without risk of any error, as to their being the subjects of inherited syphilis. I attended them both about twenty years ago, for severe attacks of symmetrical interstitial keratitis. I knew their father, and obtained his history, and the cases are, I believe, published amongst my earlier records of interstitial keratitis. They both of them had severe attacks, and, if I remember rightly, exactly at the same time, one of them being 9, and the other 11 years of age. From that time to this, with the exception that one of them, the elder, had a threatening of recurrence of inflammation in one cornea two or three years ago, they have neither of them suffered anything. Their parents being dead, they have been earning their livelihood as teachers of music. Such then being the facts, it is of great interest for us to note how very similar has been the progress of events in the two sisters, and further to observe how well they illustrate the general course in the subjects of inherited taint. They are exceptional in that the physiognomy and teeth have received so little of the characteristic stamp, but this exemption is by no means unusual. The absence of enamel-defects in the teeth enables us to assume almost with certainty, that they did not take mercury, and the narrow ridge of the nose permits us to assert with confidence that they did not suffer from snuffles. Probably, in infancy they had no symptoms and no treatment. This, I believe, is by no means unusual. The first symptom which revealed the hitherto latent taint was the attack of keratitis. If they had come under the care of any sceptic, for the treatment of the latter, they might easily have been quoted as cases in proof that this keratitis is not necessarily the result of syphilis, for the teeth of only one of the two were the sole corroborative conditions, and their malformations were so slight that, although for myself quite typical, they might easily be denied. But now observe how, at the end of twenty years, corroboration of the diagnosis comes. We have now a well characterised muscular gumma, one of the rarest and at the same time one of the most definite of the forms of tertiary syphilis. It

is rare in acquired syphilis, it is yet more rare in the inherited form. Someone may perhaps suggest that there is a fallacy, and that this young woman may have suffered from acquired syphilis also. I feel justified in putting such a suspicion aside, for I have known the sisters from their childhood. They have had free access to me in all their ailments, and I feel sure that I should have been made acquainted with it had anything of that sort happened. They have besides, been leading most meritorious lives, and are, I believe, above suspicion. We may note too that the gumma is an interstitial one, and is not attended by any disease of the mucous membrane indicative of acquired syphilis.

I may take occasion to remark in connection with these cases, as I have often done before, that the phenomena of tertiary syphilis, so well known in connection with the acquired disease, are scarcely ever met with in the inherited form. Although the inflammations of the cornea and of the middle ear, which are common in the latter, but seldom occur before the tenth year, and may be delayed as late as the thirtieth and even the fortieth, yet they have no analogies with the true tertiary symptoms. They are almost always symmetrical, always, unless destructive, transitory, and they scarcely ever show any tendency to relapse. Clearly, I think, they are parts of the secondary group which, owing probably to some law of structural development, are delayed. This view is confirmed by the fact that if parallel inflammations of the eye and ear occur in the acquired disease (they are rare), they are always amongst the secondary symptoms and not tertiary. The forms of tertiary disease which I assert that the subjects of inherited taint are usually free from, include all forms of skin disease, more particularly the syphilitic lupus, so common in acquired and never seen in inherited disease ; next, muscular gummata, gummata of the meninges, chronic ulcers on the leg, and the aggressive forms of cerebrospinal disease, such as tabes and general paralysis. I had almost included bone diseases in this list, and, had I done so, I believe I should be right, although it might need some ingenuity to convince others of the fact. The subjects of inherited syphilis are very prone to bone disease, and often show severe forms of it : but it occurs to them with very considerable differences from what we observe in the acquired disease. In the latter it is often very late, say after ten or twenty years of good health, and it does not usually affect many bones at the same time.

In the subjects of the inherited disease, on the contrary, it is almost invariably multiple, and often symmetrical, and it is met with in early youth, and is transitory. We never meet with bone disease from inherited syphilis in adults. Thus you will see that I suggest that the periostitis of congenital syphilis, so frequent and so characteristic, ranks rather with the keratitis and otitis as a late phenomenon of the secondary stage rather than as being truly tertiary. In the patient whom I have brought before you to-day we have a gumma of the tongue, unsymmetrical, developed after many years of good health—an undeniable tertiary symptom. But it is an exception to rule, and so rare that I do not know that I have ever seen another case. The usual course of inherited syphilis is that the child in infancy has an eruption, snuffles, mucous patches, iritis, visceral affections, and multiple subacute periostitis of the long bones and skull. These under treatment will probably come to an end, if the child survive, during the first year, or at most, during two years. Subsequently at any period during early youth the child may have phagedænic ulceration of the palate or the nose, transitory, and, when cured, not tending to relapse, symmetrical keratitis and symmetrical otitis, and possibly symmetrical and chronic periostitis. All these conditions will clear away after a time, and with but rare exceptions none of them will relapse. We may perhaps with plausibility invoke the fact of more rapid metabolism in early life as explanatory of the circumstance that the system appears to free itself wholly, or almost wholly, from the taint. Whatever may be the explanation, I feel sure that the clinical fact is as I have endeavoured to state it, and that the subjects of inherited taint, however severely they may have suffered in infancy, are usually in adult life, with the exceptions referred to, wholly free.

Psoriasis of the Nails of all the Fingers and Toes.

A sturdy little boy, florid, and apparently in the best of health, was brought by Dr. Nottingham on account of disease of his nails. The disease affected the nails of all his digits, of feet as well as hands, and it was exactly alike in all. Most of the nails of his fingers were, with the exception of a small portion close to the root, entirely destroyed,

and the nail-bed, in a dry and hardened condition, laid bare. What remained of the nail was tilted upwards by the accumulation of epidermis under it. The nail itself however, although broken, was not thickened, and its upper surface, in some instances at least, remained smooth. On the toes, the nails were not so much broken away, but were lifted up, some of them to an angle of 45° by the accumulation of dry, hard epidermis under them. The ends of the digits were not in the least inflamed, and the conditions had not given the boy any discomfort. In the search for any accompanying skin disease, some very minute spots like psoriasis were found on the right elbow. At the fronts of both knees, and of both ankles were large ill-circumscribed patches, which were dry and rough, but more like those of ichthyosis, or those consequent on much kneeling, than like psoriasis. The patches had not attracted much attention, and might have been present for a long time, but the boy's father was certain that the disease of the nails dated back only about nine months.

Comments. The symmetry and multiplicity of the nail disease in this case, marks it as beyond doubt due to some constitutional peculiarity. Anatomically it is a chronic inflammation of the nail-bed, and the destruction of the nail itself is secondary. In these two features, the case corresponds with what we know as "psoriasis of the nails." Such, I have little doubt, is its true nature, but it is an exceptional form, and I have seen exceedingly few exactly like it. In common psoriasis of the nail, and by this term I mean that disease of the nail which attends psoriasis of the body, the nail-bed is first affected where the disease begins, at its free border, or at its sides. But we seldom see it advance to such a condition as we have in this patient, and it is not often that the nail is much lifted up. Psoriasis of the nails is very often coincident with psoriasis of the skin, but it is often seen without it, and in some instances, it precedes its development. That it is really of the same nature is usually well proved by the fact that the remedy that cures the one, cures the other; that remedy being, of course, arsenic. I know of nothing else that does any good in these nail cases, and it scarcely ever fails. In the boy, whom you have just seen, we must note that there was no typical psoriasis, yet there certainly was a modification of it ; and he is precisely of the age at which it is likely to begin. Arsenic is clearly the remedy for him.

As an example of psoriasis of the nails without disease of the skin, and an instance in proof of the efficacy of arsenic, I may mention the case of a lady of title, who was sent to me by Sir William Roberts six years ago, on account of psoriasis of her finger nails. The condition was conspicuous, and to her great chagrin, prevented her from going into society. I prescribed arsenic, and we pushed it until it definitely disagreed, and at the same time, restored her nails to a healthy condition. I had not seen her for five years, when she unexpectedly came to me the other day for a wholly different matter, and I had an opportunity of seeing that her nails remained quite well. She told me that during the whole of this long period she had been continuing the arsenic, and assured me that if she ever left it off for a week or two, her nails were certain to begin to loosen, and break at their edges. She has taken it of late in only moderate doses, and had experienced no inconvenience from it. Her age is now 42. She has never from first to last suffered from any form of skin disease.

Herpetiform Morphœa in Symmetrical Zones.

A girl of 8, thin, but in good health, was brought by Dr. Mack, of St. Paul's Road, to show a most interesting condition of symmetrical ivory-patch-morphœa. The disease was arranged on the fronts of the thighs in patches which curved downwards, being almost crescentic in shape. They were of a dull ivory white, and attended by but little thickening. On the sides of the knees —both inside and outside—there were smaller patches quite defined, but attended by still less of thickening. There was but little of the induration of the skin which sometimes attends these cases. The girl was fully stripped, and no patches could be found elsewhere.

Comments. I claim this case as one of herpetiform morphœa or sclerodermia, although, as you have seen, it is almost exactly symmetrical. This symmetry does not in the least interfere with our creed as to its nerve-origin. It is simply an example of a symmetrical nerve-affection, just as we might have symmetrical herpes. Morphœa, which is exactly like herpes in its being located by nerves, differs from it in this, that it is very frequently bilateral, whilst herpes is scarcely ever so. We have some very remarkable cases of morphœa in zones, *i.e.* in which bilateral patches meet each

other in the middle line, and are separated from other patches above and below by belts of healthy skin. Surely, nothing could be more conclusive as to nerve causation than such an arrangement. It is seldom that we find any two cases exactly alike, and I never before saw the patches arranged precisely as they are in this child. The case, however, offers no exception to rule, but rather conforms to it. The disease is always met with, as in this instance, in healthy patients, and without any obvious or exciting cause. Slowly, in the course of years, these ivory patches will disappear, and five years hence, excepting, perhaps, that the skin will look a little shrivelled, brown, and thin, very probably there will be nothing to be seen. The prognosis, however, is not so good in all cases, for in some the subcutaneous cellular tissue, as well as the skin, is affected, and a good deal of contraction and rigidity may ensue. In some the muscles and even the bones themselves are involved, and if the patient be young arrest of growth may ensue. It is this malady, occurring in young persons, which gives us the condition which has attracted so much attention under the name of " hemiatrophy of the face."

An Example of Cure of Lupus.

The subject of this case was a young woman of clear skin, and of very delicate aspect. The tip of her nose and the adjacent parts of its sides were in a condition of thin white scar, perfectly sound at its edges, and everywhere free from the slightest appearance of lupus growth. There was scarcely any notching of the end or alæ, and the disfigurement was exceedingly slight.

Comments. I have brought this patient here in order that I may vaunt my cure. We get many patients with lupus almost well, but it is not in many that we can assert that the cure is complete. I saw this patient repeatedly during two years for lupus vulgaris of the end of her nose. She was under the care of Dr. Rogers, of Berners Street, who used to bring her to me whenever any little tubercle showed itself. The means of cure consisted in the repeated application of the caustic acid nitrate of mercury, and the chief credit is due to the patient herself, who always cheerfully submitted to the application. It is more than two years since anything was done, so that the cure has fairly stood the test of time. Let me insist that the all-important thing in the cure of lupus is

to get rid of the infective material which is its means of growth. You must eradicate the last little bit, just as a farmer would, if he could, pull up the last thistle in his field, knowing that if he did this there could be no more seed sown. If this be done in the case of lupus, and if the scar be absolutely and everywhere sound, you may fairly anticipate that there will be no relapse, for, although the disease is in a sense constitutional, it is, in a yet more important one, local. The constitutional element in our patient's case is represented by a strong family history of tuberculosis. She herself has been suspected of phthisis, and several of her nearest relatives have died of it. Although the cure is apparently so complete, and has lasted so long, yet I may quite admit that it is possible that some germinal matter may have been left behind. If so, however, it is not in an active state, and it will have to wait till some failure in general health or local degradation of nutrition may give it another chance. In two cases I have known patients who, at the age of our patient, had been equally well cured of lupus, and who had remained perfectly free for more than thirty years, experience a relapse at the age of 60.

Sodium Benzoate in Inflammations of the Upper Air Passages.

—Liegos calls attention (*Deut. Med. Wochen.*) again to the value of sodium benzoate in acute inflammations of the upper air passages—pharyngitis, laryngitis, and bronchitis. Under its use the symptoms subside in a few days. The daily dose for a child is 75 grains ; for an adult, 150 to 225 grains. In diphtheria the drug proved useless.

It is also useful in the uric acid diathesis, since it changes the insoluble urates into hippuric acid, which is easily excreted. It is a valuable cholagogue, and as such may be prescribed as follows :

R Sodii benzoatis
   Sodii salicylatis
   Pulv. rhei radicis   āā ℨiss
   Pulv. nucis vom.   ... ℨss
M.   Ft. in pil. No. xx. Sig.: One pill twice daily.

Obstinate Headache due to Neurasthenia and Chlorosis :—

R Antipyrin   ...   ...   gr.iv
  Caffeine ...   .   gr.$\frac{1}{4}$
  Cocaine hydrochloras   ...   gr.$\frac{1}{6}$
  Pulv. opii   ...   ...   gr.$\frac{1}{8}$
M.   For one cachet.—(*La Med. Mod.*)

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON SOME

CASES OF CYSTITIS, ESPECIALLY WITH REFERENCE TO ITS TREATMENT BY POSTURE.

Delivered at St. George's Hospital, Feb. 12, 1895, by

WILLIAM H. BENNETT, F.R.C.S.,

Surgeon to the Hospital, and Examiner in Surgery at the University of Cambridge.

GENTLEMEN,—The affection to which I wish to call attention to-day is a common one in the broad sense of the word; it is common in hospital work, and also in ordinary practice. This for obvious reasons makes its consideration all the more valuable.

Cystitis is an interesting subject, and there are some points in connection with it which, generally speaking, I do not think seem to have sufficient attention devoted to them.

By way of introduction, let me refer to a patient of mine, a woman in Wright's Ward, whom I mentioned the other day in illustration of a different matter, and upon whom I performed the operation of median colotomy on January 10th. She did admirably well after the operation, and nothing of any kind happened to cause her discomfort or distress until about ten days subsequently. I was then told she had developed cystitis; that is to say, she was suffering some pain on passing water, and her urine was thick and unpleasant to the smell. It was therefore suggested to me that the time had come for the bladder to be washed out, which, as you know, is the common practice in the treatment of cystitis. That treatment, however, did not exactly suit my views in this instance. Therefore, instead of treating this case of subacute cystitis by washings, etc., I insisted upon the patient, although she was still rather ill, being sat up in bed so that her body should be in an upright position. This was done, at first with some little difficulty, because she was so weak and faint; shortly afterwards she was got into a chair, and so she sat about the ward. As

her position was altered from the horizontal to the upright, the cystitis began to get rapidly better, and soon disappeared without the use of drugs or washings or any other treatment, simply because she had been placed in an upright position. This is a point upon which I wish particularly to insist—I will explain my reason presently.

Here is another instance, a woman upon whom I operated for strangulated hernia. She had always been perfectly well, till the time of the strangulation of the hernia. After the operation she was a good deal collapsed, but progressed favourably. On the third day, earlier than in the case just mentioned, she also developed symptoms of subacute cystitis; there was some discomfort on passing water, as well as some thickness of the urine itself, with a little offensive smell. In that case, which was not under my complete control, I heard nothing of this cystitis until a week after its commencement. In the meantime the bladder had been emptied by a catheter night and morning, and had been washed out with various antiseptic solutions; in addition to this, the patient had been given certain drugs which are supposed to modify the state of the urine in such cases, but in this particular instance no good result whatever followed. As soon as the washing was discontinued the urine resumed its unpleasant condition, and the cystitis returned. When I saw the patient I knew at once the cause of the symptoms, and instead of continuing these drugs and washings, all I did was to get the patient up out of her bed and seat her in a chair. Within a week from the time of commencing this simple proceeding, although she had been affected by the cystitis for something like eight or nine days previously, the disease had entirely disappeared; whereas, in spite of the washings and drugs, which had been continued during the whole week before I saw her, no improvement followed.

These are two excellent cases to show a very definite fact, which some of you may not understand. It is found, as a matter of experience, that the chronic and subacute cystitis most commonly met with is mainly due to spontaneous changes occurring in the retained urine; that is elementary and undeniable. Therefore in all these cases of

cystitis, chronic or subacute, the first thing to ascertain is whether there is any urine habitually retained in the bladder. If there be retained urine, the next thing which requires to be elucidated is the reason for the retention. I am of course not referring to acute complete retention, in which no urine is passed at all excepting that which dribbles away—the overflow.

The cases I am now dealing with are those in which the bladder can be very nearly emptied, but not completely so. Now, what is the reason which gives rise in some of these cases to this partial retention of urine in patients who present at first no objective condition likely to produce the trouble? Before going further, let me remind you of this fact, that if urine is retained in this way—if there is what is called residual urine in the bladder, it is only a question of time as to when that urine will decompose, and, if the urine decomposes, it will give rise to what we call chronic or subacute cystitis.

You will have noticed that both the patients I have mentioned are women, and there is a very remarkable fact with regard to the female bladder, which has a distinct bearing on practice. You may take it to be true that in the very large majority of women it is quite impossible for the subject to completely empty the bladder whilst lying perfectly flat on the back. Therefore, if women, on account of illness, are placed flat on the back sufficiently long, you will find that a certain amount of urine is almost certainly retained, and, in consequence of such retention, changes are prone to take place in the urine which give rise to symptoms of cystitis.

The question naturally follows: Why cannot a woman empty her bladder whilst she lies in the flat position, although a man in health, under similar circumstances, can do so unless he be the subject of prostatic enlargement? for under ordinary conditions a man up to 50 or 55 years of age, whose vesical organs are in a sound state, can empty the bladder as he lies flat on his back. The answer to this question is quite elementary, but it is necessary for me to give certain elementary details in order presently to make my meaning clear. The adult female bladder is generally, as of course you are aware, very much larger than the male bladder; it is also much more flabby, and has far less tone. But you must not suppose that this is the case throughout life, for, in very young subjects, the male and female bladder

differ only in that the male bladder is of very slightly greater capacity than that of the female. But as women grow up, partly, no doubt, in some cases as a consequence of indolence, but in the majority of cases on account of the feeling of modesty in these matters which influences women in certain habits in these highly civilized times, the urine is retained, unnaturally, for a very long time. Owing to the simplicity of the female vesical apparatus, urine can be retained very much longer by a woman than by a man under ordinary circumstances. The well-known result of this unnatural voluntary retention is that the bladder gets more and more dilated, until its capacity may become enormous. Instead, indeed, of being a strong, healthy, muscular organ, like that of the male, it becomes, in females, especially in those who have borne children, and more particularly in those who have borne many children, little more than a flabby sack, with comparatively small inherent power of contraction, and with nothing like the tone or strength of the male bladder.

In the recumbent position the bladder cannot, as I have already stated, be completely emptied by the majority of adult females. The reason of this being that the process of evacuation is effected more by the abdominal walls and diaphragm than by the muscular tissue of the organ itself. When the subject is flat on her back, a certain portion of the bladder sinks down—is weighed down in fact by its contents, and in the dependent portion the urine collects, because the organ itself has not sufficient strength in itself to expel its contents through the urethra. No urine will flow through the urethra in the female unless it is actually *expelled*, and in this matter the female is at a disadvantage compared with the male. The male urethra being a long tubular canal, the final evacuation is assisted by a process of syphonage, and is indeed almost automatic. But this is not so in the female. In addition to the natural tendency of a flabby bladder to allow fluid to collect in its lowest part, there is another reason for accumulation in the bladder of the female, especially in the cases of women who have borne children.

As a result of child-bearing, the uterus becomes heavier and more flabby. If by chance the uterus should be involved in any disease, such as a fibroid, it of course also becomes heavier than natural. It is plain under the circumstances, that the uterus then may act as a heavy weight dragging the por-

tion of the bladder with which it is in relation back towards the rectum, making that part of the viscus which is already dependent more dependent still.* It is in this depressed part that the accumulation takes place. So that there is, first of all, the natural tendency to gravitation, and, secondly, the pulling down of the bladder by the uterus. Therefore, in a case of cystitis in such a patient who has been obliged to lie flat on the back for a long time continuously, the treatment should not be by washing out and the administration of drugs, but by placing the trunk as soon as possible in a position in which the bladder can be emptied; the cure will then follow naturally, in fact it will be automatic.

Should the patient be too ill to allow of this mechanical treatment being adopted at once, washing out the bladder with antiseptic solutions, etc., and such other steps as will ensure the cleanliness of the viscus must be employed temporarily, until the patient is strong enough to justify the position treatment. The foregoing is one of the most practical points connected with cystitis which can be brought to your attention, and, curiously enough, it is one of those which is not insisted on sufficiently, although it seems to me quite obvious.

Cases of cystitis of this kind are very common in patients who come under treatment for strangulated hernia. This is especially so when the practice of raising the foot of the bed with a view to assisting reduction is practised—a method I never employ myself as I think it useless and objectionable. The position in which a patient is thus placed tends in the female directly to retention of urine. If a woman is unable to completely evacuate her urine when lying flat on the back, still less is she likely to be able to do so when the head is lower than the pelvis. For that reason, amongst others, I have always personally been opposed to this practice, although I am aware that you see it adopted occasionally in the wards of this hospital. I have no doubt, however, that it tends to increase the liability to cystitis after herniotomy. It will have been noticed that of the two cases I have alluded to, in one the cystitis supervened after an interval of ten days, whilst in the other it supervened as soon as three days after the operation. There is a very great difference in different individuals in the susceptibility of the bladder to septic or irritating degree of influences; an important clinical point. I wish to call particular attention to the fact that in the woman of whom I have been speaking, whose

* Certain uterine displacements will produce a like effect.

cystitis supervened three days after the operation *there was a high temperature*, the outcome of some inflammatory condition connected with a piece of gut badly damaged by strangulation. Under suck circumstances patients often will have a high temperature for the first three or four days. It may be taken as an undeniable fact—that in all these cases where the temperature is high after an operation, the tendency to cystitis is greater. Hence in these two cases of mine—in the patient with the high temperature, the cystitis followed in three days; but in the other, in whom the temperature was normal, it was delayed until after the lapse of ten days. A very distinct condition, therefore, which influences the period in which changes tend to take place in retained urine is the temperature of the patient—the higher the temperature the sooner will these changes occur. If the temperature is normal the changes may be postponed for a fortnight or three weeks, possibly longer, but sooner or later they nearly always come.

The difference in the susceptibility of different bladders to septic infection or irritation is very curious. As you are aware, under ordinary circumstances the mucous membrane of the bladder hardly absorbs at all. You may inject, *e.g.*, a large quantity of a very strong solution of opium without any effect whatever, either good or bad. A healthy bladder will also often retain for long periods a foreign body without any inconvenience at all, provided that no highly septic material is introduced at the same time. Tolerance of this kind varies remarkably in different subjects. The two following cases will illustrate this point :—

The first patient, a man 65 years of age, was the subject of enlarged prostate, and was in the habit of emptying his bladder entirely by the use of a catheter. He was a casual man, partly on account of cerebral indolence, the result of an ancient "seizure," and partly because he was naturally so constituted. He was not at all careful in the cleansing of his catheters. He sometimes used the same instrument repeatedly without even dipping it in water, or making any other attempt at cleaning it. On one occasion, whilst using the catheter, which was of soft rubber, the distal end passed into the meatus out of sight; he therefore seized the glans penis and pulled it forward, unfortunately without grasping the catheter. The result of this was that the catheter passed further down the urethra. Curiously enough this did not agitate the patient, in fact he took no notice of it,

and when the time came for emptying the bladder again he merely used another catheter, which, if the other instrument were not already in the bladder, must have effectually pushed it in. It must have been nearly a month after this happened when he told me, in quite a matter-of-fact sort of way, that he had lost his catheter in his bladder. At first I did not believe him, but a little later I saw him again, on account of some difficulty which he had experienced in passing a catheter. I examined the penis, and found some hard substance far back in it, which led me to think that the story of the lost catheter might be true, and so I found it to be. I extracted the instrument without difficulty. This catheter had been in the bladder for two months; it was coated with phosphatic material, and in any ordinary bladder would probably have given rise to a considerable amount of irritation. Yet the only inconvenience felt was when the end of the catheter entered the urethra, and blocked the passage. That is an extreme instance of bladder tolerance.

Here is a case of an opposite kind :—A man was brought into the Belgrave Ward, and was said to have broken, twenty-four hours previously, a bougie in his bladder, while using it as he was accustomed to do for the dilatation of a stricture. He was a man of very careful habits, and always used a clean instrument—certainly those he had with him were exceptionally clean. This patient upon admission had acute cystitis. His urine was practically full of pus, and his temperature was high. Fortunately I was able to detect and immediately remove this small piece of bougie—it was not more than half an inch in length. Here was obviously a very sensitive bladder, for very acute cystitis supervened within a few hours of the introduction of the foreign body.

An insensitive bladder, from a therapeutical standpoint, is not always an advantage; a remark which leads me to a very practical point in the treatment of cystitis. It will be found in all ordinary cases of the affection, excluding those I have mentioned as being caused by the flat position of the patient maintained for long periods, that the more easily excited a patient's bladder is, the more amenable to treatment is any cystitis which may occur—not merely to the treatment by washing out, which, after all, is only palliative in many cases, but to effective treatment by drugs. There are certain drugs which act with remarkable rapidity, and with a considerable amount of certainty, in cases of

cystitis which come on quickly; I mean especially chronic or subacute cases which originate quickly. Here let me remind you that cases which come on rapidly are not necessarily acute. The well-known peculiarity about these cases is that the urine very rapidly acquires an offensive smell; and in some of these instances of cystitis nothing is more difficult than to correct this offensive odour. Two drugs are particularly useful in correcting the offensive smell in cases of this kind —namely, salol, and betol, the latter especially, which acts with more certainty than any other drug with which I am acquainted, in suitable cases. The dose should not be too large. A dose of 5 grains three times daily is a most useful remedy, and will, as a rule, completely correct the odour in 24 or 36 hours. Bladder washing may, and should, in the majority of cases, be employed at the same time, although in many cases the drug itself will correct all the abnormal conditions without any local treatment—a fact sometimes worth bearing in mind, for it must not be forgotten that washing out of the bladder, if not conducted by persons who have a proper respect for cleanliness, may be harmful.

A CLINICAL LECTURE
ON
THE USE OF CALCIUM CHLORIDE.

By GEORGE PARKER, M.A., M.D. Cantab.,

Assistant-Physician to the Bristol General Hospital.

GENTLEMEN,—In reviewing the literature connected with this substance, one is surprised to find how long, and for how many purposes, it has been employed with apparent success. The question arises whether it is not, after all, one of those remedies which are first vaunted for some valuable property, then proclaimed a panacea, and finally dropped out of use. But the fact appears to be that while its effects have not always been correctly connected with its real action, it is a substance which naturally plays an important part in the work of the body. It is absorbed with singular ease without change, and it produces certain definite and marked results. It has three main therapeutic uses, first as a hæmostatic, secondly as a sedative to mucous membranes, and thirdly as a tissue builder in strumous diseases in place of

hypophosphites. Indeed it has been affirmed that most calcium salts, except the lacto- and other acid phosphates are first converted into chloride by the gastric juice. In fact very little of the ordinary chalk and lime preparations are absorbed at all, but owe any efficacy they possess to local action as they pass along the intestine. Of the tribasic and neutral phosphates and the common carbamate of lime it is said that not more than five or six grains are absorbed, whatever dose is given. While the carbonate neutralizes the hydrochloric acid of the stomach, calcium chloride precipitates the organic acids, but does not interfere with the hydrochloric. Thus it exercises very little hindrance over the digestion, and enters the blood in such quantities that often 35 per cent. of the quantity taken is eliminated by the kidneys in twenty-four hours. Thus Germain See mentions that a man, in some recent experiments, took daily 4 or 5 grammes, with great relief to his dyspepsia, and that he excreted .6 and even a whole gramme of the calcium daily, the total recovered from the urine being one-eighth of that taken. The elimination of lime has been said to be in the form of the acid phosphate, but moderate doses certainly make the urine alkaline, and Lehman has recently shown that the phosphoric acid is decreased. Abel, of Johns Hopkins' University, has also discovered the curious fact that the alkalinity is due to the presence of ammonia, which is set free by the carbamate of lime excreted. It is remarkable that lime should so strangely alter the metabolism of the body as to cause the excretion of this carbamic acid, which plays an important part in the changes of the organism, and is probably an immediate precursor of urea. However, its unstable lime-salt easily breaks up in the urine even of healthy bladders into ammonia, calcium carbonate, and carbamic acid.

It is well-known that lime-salts were largely given to relieve bladder irritation until the idea arose that they might aid in the formation of calculi, which put a check to the practice. Lehman denies that there is any foundation for this theory, and adds that lime is a better solvent for uric acid than soda, and causes considerable diuresis. However, we must not forget the distinction drawn by Roberts between the solvent action of a drug in the blood and its local action on the bladder urine. The effect of lime in the latter case appears to me to be still open to discussion. Spanten, indeed, praises especially the iodate of calcium for cystitis,

and the chloride has been employed with much success by Kuchenmeister and others for decreasing the amount of albumen and producing diuresis in chronic Bright's disease.

The styptic action of calcium chloride has been shown through Professor Wright's investigations, based on the work of Hammarsten, Green, and Woolridge as to the coagulation of the blood. In 1891 Wright described the effects on animals produced by, first, the internal administration of the drug in doses equal to 4 drachms in a pint of water if given to a human being. The full influence was felt in from half to three-quarters of an hour, and the results were, to say the least, remarkable. When all the veins of the neck, except the common jugular, were cut across, the blood clotted as it flowed, which stopped the hæmorrhage at once. So, too, when the central artery of the rabbit's ear was cut across. His second method was one for external use. One per cent. of calcium chloride was added to a solution of nucleo-albumen, obtained by whipping blood-clot in three volumes of water, washing the clots in a stream for not more than ten minutes, and then leaving them in five volumes of water for twenty-four hours. This solution of chloride and nucleo-albumen, or blood ferment, was applied externally, and controlled the bleeding even from large vessels. It was equally successful in an operation on the cervix uteri in the human subject where there was some difficulty in applying ligatures. He further showed that this mixture, even if it got into the vessels, would not cause intra-vascular clotting, and he injected 20 c.c. into the vessels of a rabbit with no bad results.

He next proposed the administration of calcium chloride to prevent or stop hæmorrhage in man, pointing out that large doses would be needed in sudden bleeding, since it was excreted very rapidly by the kidneys. He calculated, as I said, that the equivalent for a human being would be a dose of 4 drachms. Indeed, on working out the amounts with which he obtained such brilliant results on animals, I find that they would be even more—perhaps 5 or 6 drachms. Now, of course, this is far in excess of the doses of the drug previously used in medicine. The liq. calcii chloridi of the pharmacopœia is of the strength of 1 in 6, and the dose is set down as 15 to 50 minims only.

Thus there were difficulties in the way of carrying out to a practical end the discoveries he had made. Then, again, he proceeded to experiment

on the clotting of oxalated blood in glass vessels, and found that he got there the maximum effect with .02 per cent. of the chloride, and that excessive doses even prevented clotting. This would give as the best dose for a man weighing 10 stones about 18 grains only, so that he was led to propose much smaller doses, not only as efficient for the purpose, but actually the best. Trial was made of various amounts given internally for hæmophilia and other complaints up to 120 grains a day. Quick coagulation appears on a survey of these instances to be obtained by daily doses of half to 2 drachms, but in most of them it became abnormally slow after two or three days. Certainly in animals he 'gave only a single dose. The difficulty seems to arise when we attempt to keep the coagulation time permanently short. Indeed, after the drug had been given for a few days and then stopped, the quickness of the clotting would increase, as though aided by the elimination of the excessive dose through the kidneys. However, on analysing his cases, it seems very doubtful whether the tendency to increase the slowness of clotting after some days did not occur just as much with small doses as with large ones. With the very same amount, 30 grains t. d. s., which he noted as followed by great slowness on the third day, he obtained his best results in another case, where he reduced the clotting time from 6 minutes to 1½, during a whole week during which the trial lasted. The only factor which seemed to have much effect in aiding the object aimed at was a full diet. After meals the clotting time in normal individuals is much less than during fasting, and by a full diet he was able in a case of aneurysm to obtain the longest continued effect. Whether the test-tube experiments were or were not analogous to clinical conditions, he clearly shows that doses of 100 to 120 grains in a day do greatly increase the rate of coagulation for a time more or less prolonged. In the majority of cases what we need is temporary increase of coagulability, and his evidence from animals and human beings proves, I believe, that this is attained easily by large doses. As to the danger of these doses, I have personally found 3 or 4 drachms of the salt well diluted can be taken without inconvenience in the day. Indeed, the warning of Rabuteau that excessive doses act, like those of potash, by causing a lowered temperature and arrest of the heart's action are, I fancy, part of the theoretical results which Brunton has discredited. The question

how we may prolong this state of rapid coagulation and avoid any reactionary state is not yet clear. Possibly frequent feeding, or any means which cause leucocytosis may afford the key to it. For causing rapid clotting and cessation of hæmorrhage I have found half-drachm or drachm doses act too well in many instances to incline me to use smaller ones. Only once in a case of obscure abdominal tumour, out of many instances, did I have any complaint of palpitation, and once a slight urinary irritation appeared to follow its administration. It is, of course, difficult to say in a given case that the hæmorrhage has ceased in consequence of the drug, but I have a phthisical patient with recurrent hæmorrhages who, for the last three years, has taken it in drachm doses with considerable success, the only drawback being that the sputum is more difficult to expectorate ; and I constantly employ the remedy for similar cases and for various forms of hæmorrhage of minor severity in the out-patient room.

Some forty years ago it was thought that a cure for uterine fibroids had been found in this drug from the rapidity with which it reduced the hæmorrhage. It was imagined to cause calcification of the vessels, but we now see that it acted as a simple styptic. In menorrhagia not associated with gross local lesions, both in young women and at the menopause, it has been of frequent utility. Mayo Robson's employment of it in doses of 1½ drachms daily before operations is well known, and it has been suggested that one or more doses before parturition might prevent post partum and other hæmorrhage, which so often tax our skill and resources to the utmost. A patient with hæmorrhagic purpura was recently under my care, and after other means had been tried in vain this drug stopped the bleeding completely. It has been tried in aneurysms hitherto without success ; whether it be that we do not yet know the means of rendering its action sufficiently lasting, or whether it be that the drug has no power to induce clotting within the vessels, at least while the endothelium is intact. Nor is there reason for supposing that it will replace torsion or ligatures in surgical operations, though it may be a valuable adjunct. Still there are many cases of internal and external hæmorrhage where we have an ample field for its use. Some cases of gastric and typhoid ulceration, and especially the accidents of hæmophilia, suggest themselves. Wright reports a very severe case of the latter complaint, where, in spite

of the greatly increased coagulability of the blood which was produced by the chloride, the hæmorrhage could not be controlled until he gave inhalations containing CO_2 with complete success. This, combined with the use of chloride, appears to be the most powerful hæmostatic we possess; but for external use it is possible that his mixture of blood ferment with chloride may produce important results.

To pass on to the action of this salt as a gastric sedative. Germain See speaks very highly of all the three halogen salts of lime, as absorbed with readiness and having marked properties in dyspepsia and gastric affections. Instead of causing irritation, great relief is given by the administration of calcium chloride in cases where alkalies before food are indicated. It is best taken well diluted with a little syrup, chloroform water, and nux vomica. Two patients were recently under my care in Thomas Ward suffering from gastric ulcer. The usual treatment of peptonized milk and bismuth preparations had very little success with either. The one, a particularly chronic case, had slight symptoms of hæmorrhage, and the other suffered severely from the acute pain; but both obtained rapid relief when calcium chloride was tried. In such affections as gastric ulcer and indeed in the relief of hæmorrhage, we cannot be too careful to avoid the fallacy of *post hoc, propter hoc*, but there appears to be considerable evidence of the real efficiency of this drug.

As a substitute for hypophosphites we have the experience of Dr. Sawyer, who speaks warmly of the results he obtained in chronic phthisis by its use. Vander Corput employs it as an emulsion with cod liver oil, and Begbie wrote much of its value in the treatment of strumous glands. We have seen that its ready absorption is an important advantage in its favour, and this combined with its sedative action on the stomach renders it perhaps the best means for the administration of lime when needed for the formation of healthy tissue, whether in rachitic or phthisical patients. The doses found useful hitherto have been small ones; Dr. Sawyer gave six grains three times daily, and of course diet and other remedial measures must be attended to. In chronic or quiescent phthisis I should at the same time give creasote regularly, since not only do practical results recommend it, but Fyffe has shown that it actually does neutralize to a great extent the activity of the bacilli.

It is interesting to note the recent important

results obtained by use of Llangammarch barium waters, not only in heart affections, but also in strumous diseases and gastric irritation; and it appears that this spring contains not only barium chloride but a large quantity of calcium chloride, so that it becomes an open question whether its value does not depend quite as much on the action of the calcium as on that of the barium, at least in many of the diseases for which it has been used. The improvement effected by a course of the waters in patients suffering from glandular swellings recalls the remark of R. W. Creighton, who says that no other therapeutic agent, in his experience, produced the same good results as calcium chloride in the treatment of glandular swellings of the neck, where the glands seem massed together and of stony hardness; and in preventing suppuration.

I have tried to point out some of the uses of this substance, which seems to me to have claims to more extended employment, both as a hæmostatic, a gastric sedative, and in the treatment of tubercular glands. In the latter classes of disease, at any rate, it is not necessary to go beyond ordinary doses, while in hæmorrhage care must be used in employing larger ones. It is to be hoped, however, that before long farther investigation may show what is really the best dose, and place its administration on an exact scientific basis.

TWO CASES OF

ABDOMINAL SECTION FOR ADVANCED ECTOPIC GESTATION,

WITH

SUPPURATION OF THE SAC.

By CHARLES J. CULLINGWORTH, M.D.,

Obstetric Physician to St. Thomas's Hospital, etc.

THE two cases of which I shall speak contain many points of the highest clinical interest. Neither of them came under my observation until the child had been dead for some time and the mother had become septicæmic: so that in each instance the operation was undertaken not as a matter of choice but of urgent necessity. It is always pleasanter to have to record successes than failures, but it is

often from failures that the most valuable lessons are to be learned. On that account, and because I am strongly of opinion that every operator who publishes his cases at all should be even more particular to publish his unsuccessful cases than his successful ones, that I place these two cases on record. Hitherto my results in operations for ectopic gestation, both in the earlier and later stages, have been perhaps, on the whole, exceptionally fortunate ; but this makes it only the more incumbent on me to give publicity to fatal cases when they occur.

The main value of the following histories consists in the striking illustration they afford of the risks involved by waiting for the death of the child in these cases before operating. The fact is that the risks of operating during the life of the child are almost equalled by the risks of not operating until after its death. In the former case the patient is liable to die of hæmorrhage if we attempt to remove the placenta, and of septicæmia if we leave it to be dealt with later. In the latter case the child, from the moment of its death, is liable to undergo putrefactive changes; and we may be called upon to undertake one of the most severe operations in surgery at a time when the patient, as in the instances here recorded, has become saturated with septic infection, and is in the worst possible condition for bearing the shock of a capital operation.

It will be convenient to give the details of my cases before calling attention to the special points of interest that they present.

Case 1. E. W., a married woman, aged 33, was sent into the General Lying-in Hospital, York Road, Lambeth, under my care, on October 2nd, 1892, as a case of "missed labour." She was examined by the house-physician, Dr. Giles, under chloroform. The abdomen was enlarged to the size of a full-term pregnancy. The cervix uteri was dilated sufficiently to easily admit two fingers. The body was enlarged and, except for some soft membrane of fœtid odour, empty. Ectopic gestation was diagnosed, and on the following day, October 3rd, 1892, the patient was transferred to the ward under my charge at St. Thomas's Hospital.

Her health had been fairly good until the birth of her sixth and last child, three and a half years ago. Between that time and the commencement of her present symptoms, eight months ago, she had suffered from pains in the chest and occasional

cough, but had not been laid up or had any definite illness.

Menstruation last appeared about the end of January, 1892. Soon after that the patient began to suffer from abdominal pain and frequent attacks of vomiting. About the middle of April she took to her bed, and did not leave it for nine weeks. During this period she states that she suffered from frequent vomiting, and from attacks of severe pain in the abdomen, back, and thighs. She was only able to take a little fluid nourishment, and lost flesh considerably. After leaving her bed about the end of June she continued to have attacks of pain and to feel very ill, but was able to go about until a month ago, when she again had to go to bed. Four days before admission she thought labour was coming on.

On admission she was so ill, sallow, and emaciated, as to suggest the appearance of a patient in the last stage of phthisis or of malignant disease. There was a small bed-sore over the sacrum, surrounded by thin, unhealthy, dusky-looking skin. There were two other unhealthy sores on the legs. The temperature was 100.6°. The urine was acid, of specific gravity 1010, and contained no albumen.

The abdomen was enlarged to a size equal to that of a full-term pregnancy, and was of irregular shape. The skin was tense, and here and there the superficial veins were enlarged. The whole abdomen was extremely tender. The upper border of the swelling could be felt two inches above the umbilicus in the middle line, and rose higher on the right side than on the left. A fluid thrill could be obtained from side to side. At the most prominent part on the left side something like a fœtal limb could be distinguished. No fœtal movements could be felt, and the consistence of the mass did not alter during palpation.

There was dulness on percussion over the swelling from the pubes up to an inch below the umbilicus. The right flank was resonant, the left dull. The following measurements were taken :—

Girth at umbilicus ...	...	... 33¾	in.
Umbilicus to ens. cart.	...	... 4½	,,
,, pubes	...	... 8	,,
,, a. s. sp. r. ilium.	...	... 7½	,,
,, a. s. sp. l. ilium.	...	... 8¾	,,

The vaginal roof was depressed by a hard mass, which could also be distinctly felt through the posterior wall of the cervical canal. The uterus

was pushed forwards against the anterior abdominal wall. The cervix easily admitted two fingers. The corpus was readily explored in its entire extent. It was empty except for some extremely offensive shreds of membrane, most of which were removed.

At 2.15 p.m. on the day following her admission, she was placed under ether by Mr. White, and an incision, 4½ in. long, was made in the middle line of the abdomen, commencing just below the umbilicus. The abdominal wall showed some inflammatory thickening, and there was a good deal of venous bleeding on cutting through it. The fœtal sac was adherent to the abdominal wall, and the incision opened directly into it. A quantity of thin brownish offensive pus thereupon gushed out. The right shoulder and arm of the child presented at the opening. The hand was drawn into view for the sake of certainty, but was at once returned and the head sought for. It was found to the left and upper part of the sac, the breech lying to the right. The child, a female, was extracted by the head. It was in a macerated condition, the cuticle having become separated and rolled up into folds. (Its length was 19 inches and its weight 5 lbs. 9½ oz.) The cord (which was of a brownish colour) was divided, and the child removed. The sac was now explored, and was found to be completely shut off from the general peritoneal cavity. Its whole inner surface was of a dark colour and in a sloughing condition. Its connections were too extensive and intimate to justify any attempt at removal. The placenta lay behind and to the right, partly in the true pelvis and partly in the iliac fossa. Its separation was now proceeded with. When this had been completed the sac quickly became filled with dark blood. The aorta was thereupon compressed, and the bleeding thereby controlled until a number of bleeding points had been secured. At this stage of the operation the pulse became alarmingly feeble, and three and a half pints of warm saline solution were allowed to run slowly into a vein opened by the resident assistant surgeon, Mr. E. C. Stabb, at the right elbow. The pulse quickly improved. After an interval a further quantity of the solution, consisting of half to three-quarters of a pint, was allowed to flow in. The bleeding was taking place from the situation of the ovarian vessels, where a large vein was tied in two places by passing an aneurysm-needle beneath it and the tissues immediately surrounding it. Pus was now

seen pouring from a pouch at the right upper corner of the fœtal sac. The wall of this pouch was therefore cut away. The cavity was now thoroughly sponged; strips of iodoform gauze were packed in the pelvis; two india-rubber drainage-tubes were placed in the lower angle of the incision; and the abdominal wound was closed.

The operation lasted an hour.

The patient rallied a little from the operation and became conscious, but at twenty minutes past six in the evening she became suddenly worse and died in about five minutes.

Description of the placenta. The placenta was kidney-shaped, variable in thickness, and thin at the edges. It measured 8½ in. by 5 in., and weighed 2 lbs. The cord was inserted close to its edge.

Autopsy, twenty hours after death. The gestation-sac occupied the greater part of the abdominal cavity, and did not communicate with it. There were a few drachms of blood-stained fluid in the sac. The intestines and liver were pushed upwards. The inner surface of the sac was roughened and in a necrotic condition. The wall of the sac was about ¼ in. thick. Anteriorly, it was adherent to the abdominal wall up to a distance of three fingers' breadth above the umbilicus. Superiorly, it was firmly adherent to the intestines. It covered the uterus (which bulged into the sac) and other pelvic viscera, and extended outwards over the iliac fossa. The placental site was on the right side and extended upwards into the iliac fossa. The two ligatures included a portion of the sac wall and large vessels beneath it. On separating the sac, the left ovary and tube were found to be normal; the right ovary and tube could not be traced. The right ureter was dilated along its whole length. The uterus was enlarged; its canal measured 5 inches in length; its inner surface was covered with sloughing decidua; its wall measured an inch in thickness, and was soft and friable. The liver and spleen were enlarged. There was no evidence of peritonitis.

There were old pleuritic adhesions and some grey granulations at the apices of both lungs. There was some old pale adherent clot in the right ventricle, extending to the valves of the pulmonary artery. In the artery itself there was a dark clot, which was not adherent.

Remarks. The nature of this case had apparently not been suspected by the medical practitioner

in attendance. In the note which he addressed to the Lying-in Hospital when applying for the patient's admission, he spoke of the case as one of "missed abortion," probably a slip of the pen for "missed labour." The history obtained was very meagre and unsatisfactory, but it seems clear that the patient became alarmingly ill about the middle of the third month of her pregnancy, and was confined to bed from that time for a period of nine weeks. There can be little doubt that this attack corresponded with the rupture of the pregnant Fallopian tube and the escape of the foetus into the broad ligament. The existence of so complete and thick-walled a sac as was found at the operation, and its complete isolation from the peritoneal cavity, can scarcely be otherwise accounted for than by supposing that the development of the foetus had, from the moment of rupture, taken place extra-peritoneally, that is, in the fold of the broad ligament, and that no secondary rupture of that structure had occurred, the pregnancy being intra-ligamentous to the end. From the length of the child (19 inches), and from the size and weight of the placenta, it is evident that the child remained alive up to the eighth month, and that its death must have occurred about a month, at the most, previous to the patient's admission. Decomposition must therefore have commenced immediately and proceeded with great rapidity to have reached so advanced a stage in so short a time. The extreme emaciation of the patient was due, partly no doubt, to the continued vomiting during her pregnancy, and partly to the extensive suppuration within the sac and the septic absorption that had been going on during the month preceding her admission. The attack of intermittent pain which led the patient to summon her attendant four days previous to her admission under the impression that labour was commencing appears to have corresponded with the end of the ninth month, and the patient's interpretation of the symptoms was probably correct.

There was of course no alternative but to operate, though the condition of the patient was so desperate that one had to face the possibility of her succumbing before the operation could be concluded. But for the repeated intra-venous saline infusion there can be little doubt she would have done so, and I have to thank Mr. Stabb for his promptness and dexterity in thus averting for the time a fatal issue. Notwithstanding the ad-

vanced changes that had taken place within the sac the removal of the placenta was followed by alarming hæmorrhage. This was temporarily arrested by compression of the aorta, and ligature of the enormously enlarged ovarian vessels, which could be felt and seen running down behind the sac on the right side, completely stopped it. As there was no time to be lost in dissecting out the vessels, they were secured by passing an aneurysm-needle through the sac wall, underneath the vessels, and through the sac wall again on the other side. The dangerous proximity of the ureter was not forgotten, but the occasion was not one for excess of caution. It was interesting to note, at the autopsy, that the ligature had passed close to the ureter but had not injured it.

Case 2. M. T., a married woman, aged 43, residing at Rochester, was sent to see me by my friend, Mr. Albert Bell, on the 26th January, 1895, under the following circumstances.

The patient was married in June, 1879, at the age of 27. Early in her married life she had missed one menstrual period on three or four different occasions, but there was no evidence that this had been due to pregnancy. From 1880 to the beginning of the year 1894 she had menstruated regularly, her last normal period having commenced on February 17th, 1894, and terminated on February 20th. When the month of March passed without the catamenia appearing, the patient concluded that she was entering upon the menopause; but in April she noticed some enlargement of the abdomen and thought that she might be pregnant. About the last week in April she was suddenly seized, whilst in apparently good health and attending to her ordinary household duties, with violent pain in the abdomen. She was so ill and the abdomen was so tender that she could not be properly undressed, and remained for a fortnight on a couch in the kitchen. During the first week of the illness there was no vaginal discharge, but during the second week a discharge took place, resembling an ordinary menstrual period, except that it was more profuse than usual and accompanied by the passage of clots. She thought this might be a miscarriage, but no foetal structures were identified. By the end of the third week from the first onset of the symptoms, she was able to walk about and was soon quite well again. Since that no further discharge had taken place. The abdomen gradually increased in size, and at the end of September she felt

distinct fœtal movements. These increased in violence during the two following months, and in November the breasts became enlarged. On December 16th the patient had a few intermittent pains in the lower part of the abdomen, and a similar attack occurred on January 5th, 1895. Towards the end of December the breasts became flaccid, and the fœtal movements ceased to be felt. The abdomen now began to feel "sore," and vomiting was excited by every attempt to take solid food. Three weeks ago, for the first time since May, a slight discharge of blood commenced; this has continued more or less ever since. Thinking it might be due to placenta prævia, Mr. Bell made a vaginal examination on the 25th January, and being unable to detect anything like an os uteri, he advised the patient to come up for my opinion; this she did on the following day. The patient then looked pale, anxious, and ill. The abdomen presented a remarkable appearance, as though a full-time pregnant uterus had become tilted over to the right side, so as to be lying almost transversely. There was a tense prominent tumour occupying the lower half of the abdomen on the right side. The note over it was tympanitic. The tense swelling extended to about two inches beyond the middle line; a little below the umbilicus, and to the left of it there were little hard prominences to be felt like parts of fœtal limbs. At the extreme left side low down in the iliac fossa there was a tense cystic swelling, equal in size to a cocoanut. The note over this swelling was dull. No fœtal movements or fœtal heart-sounds could be detected. A tense swelling, of the size and shape of a distended Douglas's pouch, projected downwards in the vagina. On the right side the whole of the vaginal surface was depressed and was within easy reach of the finger, but the left and anterior fornices were drawn upwards out of the pelvis beyond reach. The os and cervix uteri could not be felt.

I came to the conclusion that the case was either an extra-uterine gestation with dead child, or an intra-uterine pregnancy, in which the uterus had been tilted over, owing to the presence of a tumour. On the whole I inclined to the former view. I expressed the opinion that in either case the proper treatment was to open the abdomen with as little delay as possible. The patient insisted on returning to Rochester, and I gave her a letter to Mr. Bell, telling him my view of the case. She returned to London the following day

(January 27th), and was admitted into St. Thomas's Hospital under my care. The temperature on admission was 101° (at midnight it had risen to 102.4°). The pulse was 128. The tongue was coated with a thick, dirty, white fur. The urine was acid, of sp. gr. 1012, and contained about $\frac{1}{30}$th of albumen and some mucus.

On January 30th Dr. Herman kindly saw the case with me, and we examined the patient under the influence of an anæsthetic. The os uteri was now found to be high up above the symphysis pubis in the middle line; it was flush with the vaginal wall, and sufficiently patulous to admit the forefinger as far as the first joint. The cervix uteri could be felt through the abdominal wall, about an inch above the symphysis. The body of the uterus could not be made out. The bladder sound passed three inches in the middle line, two inches when the point was directed to the left side, and four inches and a half when it was directed to the right. The other signs were as already described, except that the percussion note over the lower part of the abdomen, on the left side, previously dull, was now more or less resonant. The percussion note over the right side was still tympanitic.

Dr. Herman agreed with me as to the difficulty of diagnosis, and the necessity for immediate operation.

Operation. Abdominal section was performed on February 1st, at 2 p.m., Dr. Herman being present. An incision, five inches in length, was made in the median line. On opening the peritoneal cavity the uterus was seen lying immediately beneath the incision, and it was at first thought that the pregnancy was intra-uterine. On further exploration, however, it was found to be in the right broad ligament, the anterior surface of the sac being on the same plane as the anterior surface of the uterus. The cystic swelling that had been felt in the left iliac fossa, and that was thought to be a separate tumour, was now found to be continuous with the main mass. Before opening the sac, it was thought desirable to separate adhesions, which were especially numerous at the left upper corner of the swelling. During these manipulations, the sac was ruptured some distance above and to the left of the incision, and a quantity of offensive purulent fluid escaped. The abdominal incision was now enlarged a little, the sac laid freely open, and the child extracted head first. The child, a fully developed male, 20½ inches in length and weighing 6 lbs. 4 ozs.,

was swollen by the gases of putrefaction and emitted a highly offensive odour. The skin was macerated and peeling, the cranial bones were soft and movable, and the head, enormously enlarged from putrefaction, gave a tympanitic note on percussion. (This accounted for the tympanitic note over the main tumour on the right side of the abdomen.) The cord having been divided and the child removed, the placenta was next separated and removed. It weighed 2 lbs. 8 ozs. and measured 9 inches in diameter. There was no hæmorrhage. The sac and the uterus were brought up out of the wound, and it now became apparent that the sac was formed by the right broad ligament and by peritoneum stripped from the whole of the posterior surface of the uterus and from portions of the pelvic floor and walls. The membranes were separated as far as possible from their adhesions, but in Douglas's pouch a deep-lying portion of the sac had become so intimately adherent to intestine that it was necessary to leave it. The sac above this was cut away and removed with the uterus, after tying and dividing the broad ligaments and securing the uterine arteries as in the operation of abdominal hysterectomy. The patency of the cervical canal having been tested by the passing of a sound from above, the edges of the uterine stump were brought together by means of eight Lembert's sutures of fine silk. Some torn omentum was then tied and cut away, and the peritoneal cavity douched with boracic acid solution at a temperature of 110°. A glass drainage-tube was passed down into the portion of sac left attached in Douglas's pouch, and the abdominal incision was closed by means of twelve deep and two superficial sutures of silk-worm gut.

The operation occupied two hours and twenty-five minutes, and was, on the whole, well borne by the patient. Twenty minims of brandy had been injected subcutaneously during the operation.

Description of the gestation-sac and the portion of uterus removed. The portion of uterus removed measures 3 inches transversely, 1½ inches longitudinally, and an inch from before backwards. The length of the portion of the cavity included in the part removed is one inch. The anterior surface is smooth, the posterior rough and denuded of peritoneum. The broad ligament of the left side has been divided at a distance of two inches from the uterus. On tracing the right Fallopian tube from the uterus it is found that the lumen has been obliterated at a distance of half inch from

the uterine end, and no canal can be traced between that point and the gestation-sac. The right broad ligament is enormously distended and forms the greater part of the gestation-sac. The outer surface of the sac is of an opaque whitish colour, and is everywhere rough from the presence of tough shaggy adhesions. The inner surface presents a dirty, greenish-yellow appearance, the result of suppuration within the sac. The chorion can be easily stripped away from its peritoneal covering. The vessels in the placental site, recognized by its pinkish-white colour and smooth appearance, are evidently thrombosed.

The patient rallied a little in the evening of the day of operation, but the pulse remained rapid and the breathing became quick and shallow. She died at 8.25 p.m. on the following day.

Autopsy. There was found *post mortem* evidence of general peritonitis, with purulent loculi in the pelvis. The ureters were uninjured, the right somewhat dilated. Early pyæmic deposits were found in both lungs and both kidneys, especially numerous in the latter.

Remarks. The first point of interest in the foregoing case is the difficulty of diagnosis. In favour of regarding it as an ectopic gestation was the long previous sterility. It is exceedingly doubtful whether the patient had ever previously been pregnant, and it is certain that she had not been pregnant for the past fourteen years. The evidence therefore, so far as this question of sterility is concerned, was on the side of ectopic gestation, for experience shows that the occurrence of ectopic gestation is often preceded by a period of sterility of longer or shorter duration. The evidence afforded by the illness in April depended upon the view taken of the nature of that illness. It was thought at the time to be a miscarriage, occurring during an attack of peritonitis. If this view was correct it followed (1) that ectopic gestation was, to say the least, exceedingly improbable, and (2) that if an intra-uterine pregnancy now existed, it must have commenced after the month of April. But it was by no means certain that there had been a miscarriage, for no ovum, or parts of an ovum, were seen in the discharges, and the enlargement of the abdomen continued and increased. On the other hand, this three weeks' illness, if not due to miscarriage, fitted in very well with the theory of ectopic gestation, for it would then represent rupture of the pregnant

Fallopian tube between the sixth and ninth week, with partial or complete escape of the ovum into the broad ligament. The severe pain, and the not very severe hæmorrhage would be thus accounted for.

Still dealing with the history, the next question is : What was the precise import of the intermittent pains described as having occurred on the 16th of December? Supposing conception to have taken place just before the March menstrual period should have appeared, the 16th of December would represent the end of the ninth month of pregnancy, and the intermittent pains might have been occasioned by a series of uterine contractions indicative of the close of a normal gestation period. The cessation of fœtal movements and the retrogressive changes in the breasts, both dating from about this time, confirm this view—namely, that the pains felt in December were a sort of false labour.

On the whole, then, the *history* of the pregnancy favoured the ectopic view. The *physical examination* gave results which were consistent with either view, *i.e.*, ectopic pregnancy or a full-term pregnant uterus dislocated by the presence of a tumour. The dragging up out of the pelvis of the cervix and the left fornix of the vagina, the impossibility of tracing the body of the uterus, and the presence of a cystic swelling in the left iliac fossa were in favour of the latter supposition; everything else was in favour of the former.

The reason why the body of the uterus could not be mapped out became at once obvious at the operation. The anterior surface of the uterus was in the same plane as, and continuous with, the anterior surface of the distended right broad ligament, so that it was impossible to have discovered its whereabouts by palpation through the abdominal wall.
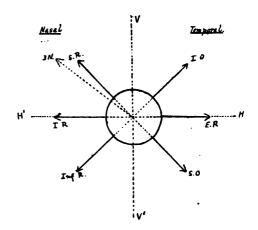
The escape of some of the putrid contents of the gestation-sac during the separation of adhesions was, though entirely unavoidable, a very unfortunate occurrence. How far it contributed to the fatal result, from direct infection of the general peritoneum, it is impossible to say. The pyæmic condition of the patient, already existing before the operation, was probably in itself sufficient to prevent recovery. And this leads me to speak of one of the most remarkable features of this remarkable case—namely, the fact of the patient's being able to go about and undertake long railway journeys with a full-term fœtus in the abdomen in so advanced a state of decomposition.

One could scarcely have imagined such a thing to be possible.

A word remains to be said as to the removal of the uterus. This was rendered necessary by the mode in which the gestation-sac had burrowed behind the uterus, stripping off the whole of its peritoneal covering posteriorly. The uterus had thus come to form part of, and to be, as it were, imbedded in the anterior covering of the sac, so that its removal became a necessary part of the operation.

The Position and Rotation of the False Image in Paralytic Diplopia, represented Diagrammatically.

By R. BRUCE FERGUSON,
M.A., M.B., B.C. Cantab., M.R.C.S., L.R.C.P.

To remember the direction of action of the ocular muscles, the position of the false image, and the rotation or torsion which this image undergoes in diplopia, is not an easy matter to every one, nor does it appear to be generally known that these and other points may be seen at a glance by aid of a simple diagram, such as the one given above.

This represents a transverse vertical section of the eye, taken in front of the equator, or, what is the same thing, it may be taken to represent the pupil of the eye. It represents either the *left* eye of a patient, as seen by an observer from the front, or the observer's own *right* eye, projected

into the field of vision, and then looked at from behind.

The diagram shows the following points :—

(i.) *Direction of action of muscles of eyeball.*

The arrows show the direction in which the pupil turns, or in which the eye will look when acted upon by the individual muscles. The four forces acting upon the globe (I.O., S.R., I.R., and Inf. R.), representing the four muscles supplied by the third nerve, may be replaced by their resultant (the dotted arrow—3 N.) Thus we see that the external rectus turns the eye outwards, the inferior oblique upwards and outwards, the superior oblique downwards and outwards, the inferior rectus downwards and inwards, and so on ; whilst the muscles supplied by the third nerve, if acting together, would turn the eye upwards and inwards in the direction indicated by the dotted arrow (3 N.). The superior rectus and inferior oblique acting together will cause the eye to look directly upwards.

(ii.) *Primary deviation of the eye in paralytic squint.*

If any muscle be paralyzed, the eye will look in the direction opposite to that in which the paralyzed muscle normally acts. If external rectus be paralyzed, the eye turns in the direction I.R., or inwards. If superior oblique be affected, the eye will deviate in the direction of S.R., or upwards and inwards. If the third nerve be paralyzed, the eye will turn in the direction opposite to that in which the dotted arrow (3 N.) points, viz., downwards and outwards.

(iii.) *Position of false image in diplopia.*

The false image is always displaced in the direction of action of the paralyzed muscle ; that is, it is displaced in the direction in which the arrow points. Thus, in paralysis of the superior oblique, the false image is displaced downwards and outwards, in reference to the position of the true image ; upwards and outwards in the case of the inferior oblique ; upwards and inwards in paralysis of the third nerve ; horizontally outwards if the external rectus be affected, and so on.

(iv.) *Quality of the diplopia.*

When the false image is displaced to the same side of the true image as the affected eye, the diplopia is said to be "homonymous"; if displaced towards the sound eye, the diplopia is "crossed." If the diagram represent the ob-

server's own *right* eye, it is obvious that paralysis of any muscle on the right (or temporal) side of the median vertical line (VV') will cause displacement of the false image to the *right*, and produce homonymous diplopia ; whilst paralysis of any of the muscles on the left (or nasal) side of the median vertical line will cause displacement of the false image to the *left*, or towards the sound eye, and the diplopia will be crossed. So paralysis of any muscle on the temporal side of the figure produces homonymous diplopia, and of any muscle on the nasal side, crossed diplopia.

(v.) *Rotation, or torsion of false image.*

Imagine the vertical line (VV') and the horizontal line (HH') to be fixed at their centres, but able to revolve, in the plane of the paper, round a central pivot.

(*a*) *Vertical images.* Let VV' revolve in the same direction as the hands of a watch, until its position approach that of I.O., Inf. R.; this gives the rotation of the false image which occurs in paralysis of the Inferior Oblique or the Inferior Rectus ; that is, the top of the image leans towards the temporal side. Now turn VV' in a direction opposite to watch-hands, viz., towards S.R., S.O., and we get the torsion of the false image which occurs in paralysis of the Superior Rectus, or the Superior Oblique ; that is, the top of the image leans towards the nasal side.

(*b*) *Horizontal images.* Proceed in a similar manner with the horizontal line (HH'), rotation of which in the direction of watch-hands, viz., towards S.O., S.R., will show the rotation of the image in paralysis of the Superior Oblique or Superior Rectus, whilst rotation of HH' against watch-hands will show the torsion of the false image in paralysis of the Inferior Oblique or Inferior Rectus.

In the case of the Internal or External Rectus there is no rotation, either for vertical or horizontal images, the displacement being simply a lateral one.

If the image looked at combine both a vertical and a horizontal line, thus ╬, then paralysis of superior oblique or superior rectus will produce ⤬ , and paralysis of inferior oblique or inferior rectus ⤢ .

To sum up the points in the case of the Superior Oblique :—

The diagram shows us that its action turns the eye downwards and outwards ; that when paralyzed, the eye deviates upwards and inwards ; that the

false image is displaced downwards and outwards with reference to the position of the true image; that the diplopia is of the homonymous variety; that if the image be a *vertical* one, the rotation will be such as to cause the top of it to lean towards the left, or " counter-clockwise " rotation; whilst if the image be a *horizontal* one, there will be " clock-wise " rotation.

THERAPEUTICAL NOTES AND FORMULÆ.

Nitrate of Silver in Phthisis.—M. Crocq has been giving this drug in doses of 1 to 5 centi-grammes ($\frac{1}{12}$ gr. to $\frac{1}{3}$ gr.) daily, in divided doses, either alone or in combination with opium, ergo-tine, gentian, or strychnine, and sums up his results as follows:—Appetite improves, cough ex-pectorations diminish in frequency and amount, respiration is easier, and digestion more complete. These results are encouraging, but M. Crocq warns that he has no proof that they are permanent.

(*Bull. de l'Acad., Roy. de Med. de Belg.*)

Erysipelas.—Dr. W. Baar reports most favour-able results in the treatment of erysipelas by means of subcutaneous injections of pilocarpin. He gives 1 centigram (about $\frac{1}{6}$ grain) as a first injection, followed by three subsequent ones at intervals of two, six, two hours respectively after one another.—(*Ther. Gazette.*)

Constipation in Young Babies.—Dr. Cahen Brach, of Frankfort, recommends cod-liver oil as the best laxative in suckling children; the addition of thirty to sixty grains of milk sugar is also of assistance. Rhubarb, manna, casc. sagrada, and other laxatives must, on the other hand, be avoided. Gentle massage of the child's abdomen is, however, perhaps at once the simplest and the best means of overcoming this troublesome con-dition, it must be used two or three times a day for some five to ten minutes at a time.

(*Zeitschr. fur Landpr.*)

Night-sweats of Phthisis.—Ergot, powdered, one or two gr.v doses before bed-time, or diluted extract of ergot subcutaneously injected.

(*Deut. Med. Woch.*)

Chloroform Anæsthesia.—Dr. P. Rosenberg asserts that the dangers arise from reflex irritation through the sensory fibres of the fifth nerve (apart, of course, from overdosing and carelessness), and recommends that the nasal and oral mucous membrane should be rendered anæsthetic by cocain before the inhalation of chloroform is commenced.—(*Berl. Med. Gesell.*)

Catarrh, Gastric and Intestinal. — Dr. Golines has tried papaïn in three to five grain doses in this trouble with excellent results, especially in cases that have arisen after influenza associated with loss of appetite; he states that it is also useful in chronic dyspepsia and even in cases of gastric dilatation.—(*Reichs. Med. Anzeig.*)

Chronic Gonorrhœa :—

R̶   Tannin   }
    Iodoform   }   ...   āā   gr.xxx
    Glycerin ...   .   ʒj
    Aqua Rosæ   ...   ...   ʒiv
M.   Fiat injectio.

(*Monatsch. Makt. Derm.*)

REVIEWS.

Pulse Gauging by Radial Measurement and Pulse Pressure. By GEORGE OLIVER, M.D. (Lewis, London.)

Published at 3s. 6d.

This little book contains an account of two new instruments, "the arteriometer" and the "pulse pressure guage," and the results to be obtained by them. After reading it we feel a little sceptical as to any clinical value in the instruments, because they do not seem to be able to give information which cannot be obtained by other methods more easily applied; as, however, Dr. Oliver claims for them that they hinted to him the possibility of long-past syphilis in the absence of any other in-dications we must withhold a final judgment on their utility for the present, and merely say that the instruments, their application, and the deduc-tions from their results are very clearly described. With the present outcry of the heads of the pro-fession against advertising, we could have wished that mention of Harrogate had been a little less prominent.

Diphtheria and its Treatment. By BROWN-LOW H. MARTIN. (Balliere, Tindall, & Cox.)   *Published at 2s.*

This is merely a short pamphlet describing Dr. Martin's treatment of the malady by means of insufflations of sulphite of magnesium. To quote his own words, the remedy is "safe, simple, efficient, and a perfect bactericide in diphtheria, and, moreover, being in the form of a very fine and sparingly soluble powder, it is during inspiration drawn into the larynx and bronchi, acting when other remedies will probably fail." Dr. Martin claims that he has not lost a single case treated thus for several years. For ourselves we can only say that if this statement be even reasonably near the truth antitoxin may be safely placed in the background.

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The Difficulties of Prognosis in Insanity. By H. SUTHERLAND, M.D. (Bale & Sons.)

This is a reprint of a paper read by Dr. Sutherland before the West London Medico-Chirurgical Society. Age is the first guide given, but here many exceptions must be made; the form of insanity affects the prognosis more deeply, epilepsy as a concomitant being especially bad omen; periodicity in attacks banishes hope of cure; homicidal insanity Dr. Sutherland considers always incurable; sunstroke as exciting cause is of bad augury; the prognosis in alcoholic insanity is entirely a question of acuteness,—acute cases do well, chronic ones badly; such are a few of the hints Dr. Sutherland gives us. The paper is well worth perusal by all those who may be called upon to deal with cases of mental disease.

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The Medical Annual. 13th Year. (Wright & Co., Bristol.)   *Published at 7s. 6d.*

All annual *resumés* must bear a certain resemblance to one another, and the reviewer's task is confined to estimating the judiciousness or otherwise of the selections made from the dreadful mass of undigested crudities which are poured forth by the medical press every year. The names of Hurry Fenwick, Colcott Fox, Greville Macdonald, Hector Mackenzie, Mayo Robson, Rugger, Saundby, and others, alone will give a

guarantee that some portions of the work have been carefully and conscientiously performed, and the book as a whole is certainly good. At the same time we would remark that nitrite of amyl and iodide of potassium are no new remedies for lowering blood-pressure. Angeo-neuroses are well handled by W. Ramsay Smith. The article on cancer might profitably have been reduced one half. Simeon Snell's article on eyesight in children, with its accompanying figures, is perhaps one of the best in the book, and deserving of separate publication. We fancied we had heard of the stem pessary before the year 1894. Should the presence of "the third blood corpuscle" prove itself pathognomonic of tubercle, it will be a valuable addition to our means of diagnosis. Such are a few of the good and bad points of the book, and if on the thirteenth annual appearance we might offer a suggestion to the responsible editors, it would be, make the book smaller, admitting nothing but what is really new and of approved utility; thus the busy practitioner may be spared some of the wading through pages of what is already familiar to find a line of useful novelty.

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Hand-book of Diseases of the Eye, and their Treatment. By HENRY R. SWANZY. (5th Edition. (Lewis, London.)   *Published at 10s. 6d.*

That a book has reached its fifth edition in a comparatively short time is sufficient proof that it is appreciated by those for whom it is written, and in the present instance we can well understand why students appreciate Swanzy's hand-book. All difficult points of practical use are most clearly explained, the woodcuts are good and correct, the pages are not overburdened with recondite speculation, interesting enough to the specialist but not required by the student. We may refer to p. 450 *et seq.*, where amblyopia and amaurosis are discussed, as an excellent example of clear writing, and the figure on p. 454 is superior to the somewhat similar one (showing the tract of visual fibres) usually found in text-books. An excellent feature, from a student's point of view, is the presence in all cases of the Greek words from which so large a part of ophthalmic terminology is derived. An excellent and accurate index completes a work which we can highly recommend as sound in principle and complete for ordinary practice.

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

A CASE OF PRIMARY CANCER OF THE GALL-BLADDER.

By J. BURNEY YEO, M.D., F.R.C.P.,

Physician to King's College Hospital, etc.

GENTLEMEN,—There has been recently in the hospital, under my care, in the Twining Ward, a case of persistent jaundice, which ended fatally on the 27th of February last. As the clinical history of this case was completed by a post-mortem examination of the body it will, I think, be instructive and profitable to you to attend carefully to the brief account of this case which I now propose to lay before you.

The patient was a female, 54 years of age, and she was admitted on the 14th of February of this year, complaining of pain in the epigastric region, vomiting, and jaundice. ·

She gave us to understand that, except for occasional bilious attacks, with constipation and loss of appetite, she had always had good health up to the onset of the present illness, which began so recently as seven or eight weeks before her admission.

The *first sign* of loss of health which she said she noticed was the occurrence of jaundice. The yellow staining of the skin by bile pigment was the first symptom to appear, and it was then unaccompanied by any other.

This yellow coloration of the skin grew deeper and deeper. But she had 'no pain or other notable disturbance of health, unless it was that she grew thinner and more feeble day by day.

Quite recently, however, she had complained of pain and discomfort in the epigastric region coming on after food. She stated that the pain was of a dull, aching character, and that it was relieved by vomiting, which was of frequent occurrence, and always after taking food. She said she felt the pains also in the back, between the shoulders. She also stated that the vomited matters had been of a *dark brown colour*, but this statement appears

to have been given in answer to a *suggestive* question, and would seem to be of doubtful accuracy. I would just remark *en passant* that it is of the greatest importance, in the examination of patients, not to put questions in a *suggestive* form.

I find in the notes of this case that the patient is stated to have described the matter vomited as "*usually dark coffee-coloured*." As no such vomit ever appeared after her admission, I think we must conclude that this was a suggestion.

Another statement noted as made by the patient was doubtless not strictly accurate, but it was, I can readily believe, a genuine impression, and one that was not uninstructive. She said she vomited about twice as much as she took. That the patient should have such an impression pointed to the probable existence of some obstruction at the outlet of the stomach, and probably also to a certain degree of consequent dilatation of that organ. The prolonged retention of food in the stomach, and its accumulation there, would lead occasionally to such copious vomiting as to give this erroneous impression to the patient.

This patient's history, then, was briefly this: she had been well up to six or seven weeks before admission, then she noticed she was getting yellow, and this yellowness deepened; at the same time she began to get weaker and thinner, and this emaciation and debility had progressed rapidly; recently she had had pains in the epigastrium, with profuse vomiting, which relieved the pain. That briefly was her history. Negatively, I may add, that she had never had anything in the shape of a paroxysmal attack of pain resembling an attack of *biliary colic*.

On her admission it was noted that she was very thin and feeble; that the whole surface of the body and the conjunctivæ were deeply bile-stained, so as to be of a greenish-yellow colour: the urine also was almost black from the presence of bile pigment; the bowels were rather constipated, and there was an absence of bile in the motions, which were white or clay coloured. The pulse was 88, small, but regular, and the radial arteries were thickened and tortuous. The tongue was clean, but dry, red, and fissured. The thoracic organs were normal.

We may note here that the pulse was not lowered in frequency as is common in jaundice, but somewhat quickened. It may have been slowed previously, and the rapidity was probably due to the febrile state that, as we shall see, was imminent. On examining the abdomen we first noted that there was no obvious distension—no notable appearance of abdominal tumour. The liver dulness was very slightly, if at all, increased. The extent of stomach resonance was also only slightly increased.

Just, however, to the right of the epigastric region, and corresponding to the situation of the *gall-bladder* and the pylorus, was felt a distinct hard mass—not large—about the size of a small pear or lemon, but of no very definite shape. It was hard and resistant, and painful when pressed upon. It clearly corresponded in situation with the gall-bladder, the pylorus, and the head of the pancreas, and was doubtless, in part, a malignant growth. The persistence and increasing intensity of the jaundice, and the very rapid emaciation and loss of strength, as well as the absence of paroxysmal attacks of biliary colic, pointed to the presence of a malignant growth compressing the bile ducts from without, as the probable cause of persistent and intense jaundice. The symptoms of pyloric obstruction, the pain and copious vomiting after food, might have been thought to point to the pylorus as the seat of the growth. But the history of the case made such a supposition untenable.

The occurrence of jaundice and progressive emaciation had preceded by several weeks the onset of gastric symptoms, from which we were justified in concluding that the growth which had caused the jaundice, from compression of the bile ducts, had subsequently come to press upon the pylorus, and that the pyloric obstruction was secondary to the biliary obstruction. Where, then, was the growth located? Was it in the head of the pancreas? as in a case I not long ago brought under your notice of a somewhat similar course and character.* The hard mass seemed too superficial and rather too high up for the pancreas, and we concluded that it was most probably in the gall-bladder itself.

The patient continued to vomit all her food and to get thinner and weaker, but complained of little or no pain, unless the mass I have spoken of was

* See *Clinical Journal*, April 18, 1894, "A Case of Obstructive Jaundice from Malignant Disease."

handled. On one occasion, the day after her admission, she vomited as much as 48 ozs.; but with more careful feeding she did not vomit more than 10 ozs. at a time. She was at first perfectly clear and intelligent, but after she had been in the hospital about a week she began to show signs of cholæmia, the vomiting ceased, she became very drowsy, apathetic, and incoherent, and was delirious at night; coincidently, her temperature, which had ranged from half a degree to a degree and a half above normal, rose to between 103° and 104°; diarrhœa at the same time set in, and she sank rapidly. She died on the 27th February, only thirteen days after her admission, and two months after the onset of the illness.

On *post-mortem* examination the liver was found to be slightly enlarged and deeply bile-stained from distension of the bile-ducts. The gall-bladder was small and contracted, and contained no bile. Its walls, however, were much thickened, and were contracted around several gall-stones of medium size—one, however, was nearly as large as a hazelnut—they were dark-coloured and facetted. The external wall of the gall-bladder was fibrous, but the inner mucous lining was thickened by malignant disease, and where the gall-bladder was adherent to the liver the growth had invaded the adjacent part of that organ to some extent. In the portal fissure, and surrounding the common duct and the pyloric region of the stomach, there was much dense fibrous tissue, of inflammatory origin, which compressed the bile-ducts and the pylorus. There was no dilatation of the common bile-duct, but close to the duodenal orifice there was a small gall-stone, not large enough, however, to obstruct the canal. The pylorus and the head of the pancreas were normal. The stomach was somewhat dilated.

There are certain points of interest to be noted in this case.

In the first place it ran an unusually rapid course. There was nothing in the aspect or history of this case on admission to lead us to anticipate so early a fatal result. She died of cholæmia, of bile-poisoning, the characteristic symptoms of which appeared during the last week of her illness; these were a moderate degree of fever, with dry tongue, quickened pulse, diarrhœa, extreme drowsiness, a quiet delirium, and finally coma.

But it is common, in cases of persistent obstructive jaundice, to observe for a long time a far more intense discoloration of the skin than we had

in this case, without the appearance of these rapidly fatal symptoms of cholæmia. A case of obstructive jaundice from cancer in the head of the pancreas, to which I called your attention last summer, was in the hospital nearly four months before it ended fatally.

The rapid termination of this case may possibly have been determined by the co-existence of compression of the pylorus and the consequent inability to retain food, so that the resisting power of the organism was weakened by mal-nutrition; or the kidneys may have been inadequate to the free elimination of the more poisonous constituents of the bile; for in all cases of obstructive jaundice poisoning from cholæmia would very soon ensue were it not that the kidneys eliminate from the blood the bile elements accumulated in it. Bouchard has pointed out that the bile is nine times more poisonous than urine, and that the liver of a man may form in eight hours sufficient poison to kill him. If, then, there is any failure in the eliminating power of the kidneys in cases of this kind, death from cholæmia must rapidly ensue.

If we look at the record of the amount of urine excreted in this case we find that it was much below the average; of course on those last days of the illness, when much diarrhœa occurred and the evacuations were passed unconsciously, the quantity of urine passed could not be accurately estimated, but the daily average for the six days preceding the onset of diarrhœa was only 25 ozs.

Another interesting point in this case is the relation of the gall-stones found in the gall-bladder to the occurrence of cancer in its walls. It is said that in seven-eighths of the cases of primary cancer of the gall-bladder gall-stones have been found in it. And it is inferred that there is an etiological relation between the occurrence of cancer of the gall-bladder and the presence of these concretions; and it has been suggested that the cancer is due to irritation caused by the gall-stones, just as epithelioma of the lip can often be traced to mechanical irritation.

In this case the presence of these gall-stones in the gall-bladder seems to have had no direct connection with the jaundice. There was never, throughout this comparatively brief illness, anything like a paroxysm of biliary colic. The jaundice came on insidiously and without the occurrence of any pain, and it was only when, subsequently, the symptoms of pyloric obstruction were added to the jaundice, that anything besides the discoloration of the skin and the progressive emaciation and weakness was noticed.

We found on post-mortem examination that the gall-bladder was small and contracted; not at all enlarged. We did not expect to find any *considerable* enlargement of that organ, as there was no clinical evidence of such a condition. But we hardly expected to find it so small and contracted; and that the hard resisting mass we were able to feel in the situation of the gall-bladder was, to a great extent, formed of a "dense mass of fibrous tissue" of inflammatory origin which surrounded and compressed the bile-ducts and the pylorus, and formed adhesions between them and the malignant growth in the walls of the gall-bladder.

The compression of the bile-ducts and the consequent jaundice was not *directly* due to the cancerous growth in the gall-bladder, but was caused by the contraction of this dense mass of fibrous tissue. The malignant growth, although it had infiltrated to some extent the adjacent portion of the liver, was by no means extensive—and the symptoms produced, and the fatal result were due to the irritative growth of fibrous tissue and the adhesions and matting together, and compression of important structures which it led to.

TWO LECTURES

ON

OCULAR SYMPTOMS IN DISEASES

OF THE

BRAIN AND SPINAL CORD.

Delivered at St. Thomas's Hospital, March 1 and 11, 1895,

By J. B. LAWFORD, F.R.C.S.,

Ophthalmic Surgeon to the Hospital, and Lecturer on Ophthalmology in its Medical School.

LECTURE I.

GENTLEMEN,—In this and the following lecture, which will be the last two of the winter course, I think we may profitably devote our attention to the ocular symptoms which are met with in some diseases of the brain and spinal cord. To a considerable extent these two lectures will be recrossing of ground already traversed this session, for, as you will remember, the causal relation existing between general maladies and ocular troubles has

been frequently referred to in the lectures on the various diseases of the eye. On those occasions, however, the question has been considered especially from the ocular side, if we may so term it. To-day, and in my next lecture, I shall endeavour to take a more general view—to look at the subject not so much from the ophthalmologist's standpoint as from that of the physician.

In speaking to you I need hardly emphasize the importance to the physician, and to the general practitioner, of careful examination for ocular symptoms in many, if not all, of the cases of disease of the brain and spinal cord which come under notice. Discovery of the presence of such symptoms often proves of real service in diagnosis, and proof of their absence is sometimes of nearly equal value. In hospital practice it may, perhaps, seem to some of you unnecessary to trouble much about eye symptoms in general disease, as in cases of doubt the ophthalmic surgeon is asked to see the patient, and clear up the nebulosity, if he can. To a certain extent this is quite true, and I imagine most ophthalmic surgeons are very glad to be asked by their colleagues to see cases of general disease which may present ocular symptoms; certainly I am. But in the future, many of you may be unable to refer cases for the opinions of specialists, and I would, therefore, urge you to use every opportunity of studying the ocular complications, or the ocular manifestations of general disease.

I shall not have time in these two lectures, nor should it be necessary, to describe to you in detail the various ocular lesions which I shall have to refer to. This has already been done in the previous lectures during the session in dealing with diseases of the different parts of the eye. Nor shall I say very much as to the pathogenesis of the ocular changes to which we shall allude.

With this preamble we can pass at once to the subjects under consideration.

We shall begin with *tumours of the brain*, or, to be more correct, intra-cranial tumours, in which category can be included meningeal growths, which are not strictly cerebral tumours. *The* ocular symptom met with in intra-cranial tumour is optic neuritis, and it is met with in a large percentage of the cases. The proportion varies considerably, judging by the writings of different authors, so that it is not an easy matter to arrive at definite figures. Gowers,* whom we may consider a special author-

ity on the subject, says that in his experience about 80 per cent. of the cases of intra-cranial tumour are accompanied by optic neuritis; and in referring to this subject, he mentions that this percentage is lower than the figures given by previous writers seem to indicate. In the Ophthalmological Society's Transactions,* there is an analysis of 107 fatal cases of brain tumour, and of these 63.5 per cent. had optic neuritis. This proportion you will notice is considerably lower than that given by Gowers. On the other hand, Annuske and Reich, two German writers, in an analysis of 88 cases, state that 95 per cent. had optic neuritis, leaving only 5 per cent. in which this condition was not present. This was some years ago, however, and, as Gowers reasonably says, in the earlier days of ophthalmoscopy cases of tumour of the brain with optic neuritis were more likely to be recorded than those in which optic neuritis did not occur. Whatever the actual proportion may be, what you have to bear in mind is, that a very large proportion of cases of cerebral tumour are accompanied by optic neuritis. In tumour of the brain, optic neuritis is nearly always symmetrical, that is to say, it is found in both eyes; it is not necessarily present to the same degree in the two eyes, nor does it always affect both eyes at the same date; there may be an interval of days, or even weeks after its onset in one eye before it appears in the other. Cases of cerebral tumour have been recorded, though they are not numerous, in which optic neuritis has developed in only one eye, and at one time it was thought by some authorities that the presence of optic neuritis in one eye only in a case of intra-cranial tumour, was of distinct value in localisation of the tumour. This view, has, I think, been practically abandoned; all one can say now is that this asymmetry in optic neuritis in cases of tumour is a peculiarity which, in the present state of our knowledge, has no definite significance, and no value can be attached to it.

The next point I wish to draw your attention to is that optic neuritis does not seem to stand in any relation to the *nature* of an intra-cranial growth, or to its *size*. It is met with in every variety of new growth—sarcoma, glioma, tubercle, syphilitic growths, cysts of various kinds; and it occurs in cases in which the tumour is of comparatively small size as often as in those in which the tumour is of very considerable dimensions. On

* "Medical Ophthalmoscopy," 2nd edition, page 157.

* Edmunds and Lawford, "Ophthalmological Society's Transactions," vol. iv., 1884, page 172.

the other hand, cases have been reported in which large tumours (in one reported by Gowers, the size of the closed fist) have been unaccompanied by optic neuritis.

Has the position of the tumour any relation to the occurrence of optic neuritis? I think it has, and my opinion is based largely upon data obtained from published cases. In the analysis of the 107 fatal cases of brain tumour which I have mentioned, it was found that in 61 of the 107 instances the tumour was situated at the base of the brain, in which region the cerebellum is included. Out of that number, optic neuritis was present in 74 per cent. The tumour occurred in parts of the brain other than the base in 47 cases, and optic neuritis was present in only one half (50 per cent.) of these. This result as well as other evidence, affords some grounds for believing that optic neuritis in intra-cranial tumour has a certain, though not very definite, value as to the position of the tumour, namely, whether it is basal, or whether it is situated in some part of the brain removed from the base.

Another point I would impress upon you is this: optic neuritis of high degree affecting both nerves may be present, and exist for some considerable time without producing any symptoms which the patient refers to the visual organs; that is to say, without interfering with sight in sufficient degree to lead to complaint in this direction. This fact, and the importance of it, were pointed out some years ago by Hughlings Jackson, and it is very necessary to bear it in mind, because you may have a case, or a suspected case, of cerebral tumour, without any complaint of failure of sight by the patient, and without finding vision materially affected on examination, and this may lead you to assume that there is no optic nerve disease. But if you do so you will certainly, sooner or later, be led astray, because, as I have said, cases may, and do occur in which well-marked optic neuritis exists without defective vision.

In chronic cases of tumour of the brain, in which symptoms exist for a long time, months may elapse before neuritis occurs, and then a well-marked optic neuritis may supervene. Hence, in these cases, it is not sufficient to examine the eyes once only, when, or soon after, the patient first comes under observation, but repeated examination should be made. One remarkable case has been published by Hughlings Jackson * in which

* "Med. Times and Gazette," 1875.

a man exhibited symptoms of tumour of the brain for nine years. During the last three years of that time the eyes were repeatedly examined ophthalmoscopically, and only six weeks before death did optic neuritis supervene. In such a case, if ophthalmoscopic examination had been made only on one or two occasions in the early period, the optic neuritis would never have been discovered. Another point to bear in mind is that neuritis may occur, and then subside, leaving the sight perfect, and may recur at a later stage if the disease of the brain persists without being fatal. Several cases of such recurrence of optic neuritis in brain disease have been placed on record. A very chronic form of optic neuritis, or perhaps one should say neuritis, which is not very severe, is generally thought to indicate chronicity in the brain tumour. In cases of rapidly growing intra-cranial growths the neuritis, when it occurs, is usually severe; this rule, however, is not without exceptions. The onset of optic neuritis, simultaneously with exacerbation of the other symptoms caused by the tumour has been noted so often that it must be more than a coincidence. And, similarly, subsidence of the neuritis has been · noted in cases where the general symptoms have retrogressed—where the patient has improved.

Now, what is the significance of optic neuritis in intra-cranial tumour? In the first place, I may say at once that it has nothing like the localising value that it was at one time supposed to have. Some years ago, it was thought that the presence of optic neuritis would be found to furnish more or less exact indications of the position of the intra-cranial growth. Increase in our knowledge, however, has forced us to the conclusion that the localising value of optic neuritis is very small, and is limited to indicating whether the growth is at the base of the brain or at the vertex or other part removed from the base. But it is a very important sign of the presence of an intra-cranial tumour, indeed, sometimes it is the only reliable symptom. Its prognostic significance is also not infrequently of definite value. Its frequent development concurrently with an increase in the other symptoms due to an intra-cranial growth, enables us to say that in a case of chronic brain tumour the onset of optic neuritis constitutes an unfavourable sign in prognosis; for, in such cases, its onset shortly before death has been frequently noted. On the other hand, you must bear in mind, that the absence of optic neuritis in such

cases does not have an opposite significance, that is to say, cases of tumour of the brain of long duration, in which optic neuritis has not developed, may eventually prove rapidly fatal, and the absence of neuritis throughout does not materially influence the prognosis. The presence of optic neuritis has a distinct prognostic value, but its absence has much less value.

Now a few words as to diagnosis. There are other conditions met with which may be mistaken for intra-cranial tumour, and in which optic neuritis is present as a symptom. Given a case of headache, vomiting, and optic neuritis (three of the chief symptoms of intra-cranial tumour), are there other diseases in which this combination of symptoms occurs? The answer is that there are, and I would mention three especially, which you will probably meet with during your attendance in the out-patient rooms.

1. In certain cases of severe anæmia, chiefly in young women, double optic neuritis is met with, and these patients also suffer from headache and from sickness; in other words, they present the three symptoms which are the most common, and perhaps also the most reliable in intra-cranial tumour. In such cases the difficulty of diagnosis may be very considerable. Ophthalmoscopic examination is not in itself sufficient to enable you to give a positive opinion, but it would, or might, be of some assistance. In these cases of anæmia, the neuritis is seldom as intense as in cases of intra-cranial tumour; that is to say, there is seldom as marked swelling of the disc, and bending of the vessels; there is seldom much hæmorrhage from the retinal vessels, and defect of vision is usually slight. In intra-cranial disease very intense neuritis is often met with, with a great deal of swelling, numerous hæmorrhages from the retinal vessels, and very marked defect of vision. But, as I have already told you, all the signs and symptoms in a case of neuritis from brain disease may be slight, and therefore it would be unwise to rely on the ophthalmoscopic examination alone; it is your duty to make use of every other means which you can bring to bear, to assist your diagnosis in such a case.

2. Another group of cases in which difficulty sometimes arises, are those which are occasionally, though not very frequently, seen in the out-patient room here, and which are sent round from the medical out-patient room for an opinion as to the condition of the eyes. The patients are usually young people, often children, who are brought to the hospital on account of headache, sometimes accompanied by sickness, and with ophthalmoscopic appearances which in some instances closely simulate those of true optic neuritis. They are cases in which on examination, an error of refraction, usually hypermetropia, is found. The headache is the ocular form, sometimes spoken of as ocular cephalalgia, and it is occasionally, though not very frequently, accompanied by vomiting. Here again, are three of the leading symptoms of intra-cranial disease. The diagnosis of such cases is not usually very difficult, although the ophthalmoscopic diagnosis is sometimes far from easy.

In some instances, it is very difficult indeed to say whether the appearances observed with the ophthalmoscope are those of genuine encephalic neuritis, or changes in the optic papillæ not very unusual in young hypermetropic eyes, and dependent upon the hypermetropia, and yet you will see that the diagnosis is really most important. Such a case can frequently be distinguished from one of intra-cranial disease by paying close attention to the relation borne by the headache to the use of the eyes. With an intelligent patient, or, in the case of a child, its intelligent relatives, you will seldom have serious difficulty in obtaining a reliable history. This may reveal the fact that the patient is liable to severe headache, with occasional vomiting perhaps, after school (or after work if the patient was at work) or following a spell of reading or needlework; but, that if, through absence from school or work the eyes were not closely applied to any task, the headache was either slight, or entirely absent. In headache associated with brain tumour, although there are variations in severity, they seldom bear such relation to the use of the eyes as in the hypermetropic cases.

3. In a few cases of chronic renal disease (granular kidney) optic neuritis is met with which is indistinguishable, ophthalmoscopically, from that caused by tumour of the brain, and these patients, as you are aware, often suffer severely from headache, and not infrequently suffer from sickness also. In such instances there may not be the well-known retinal changes of Bright's disease, but in a fair proportion of cases, in addition to the papillitis, you will find changes in the retina more or less characteristic of albuminuria. In reference to this group of cases I would just mention, though it does not belong strictly to our present subject, that there are certain cases of brain

tumour in which the ophthalmoscopic appearances are very like those of renal retinitis; and I showed here a few weeks ago, when lecturing on this subject, a drawing from a patient with brain tumour, in which the ophthalmoscopic appearances were all extremely like those of chronic albuminuria. Indeed, so closely did it resemble this latter disease that the case was looked upon for some time as possibly one of chronic renal disease, in which albumen was not constantly present, and repeated examination of the urine was made in order to establish its presence or absence. Eventually the case proved to be one of cerebral tumour.

Optic nerve atrophy is sometimes met with in tumours of the brain. Simple atrophy of the optic nerve may occur from pressure of an intra-cranial tumour, but this is not at all common. In such a case the tumour is almost certainly situated in the neighbourhood of the sella turcica, pressing on the optic commissure itself, or pressing on the optic nerves just as they leave the chiasma, and such pressure may lead to atrophy with all the ophthalmoscopic appearances of what is called primary or simple atrophy. Pressure of a tumour on one optic tract very rarely gives rise to recognizable changes in the optic discs, although causing other symptoms which I shall have to mention later on. The majority of cases of intra-cranial tumour in which atrophy of optic nerves is present are those in which this condition is the result of optic neuritis—in which the neuritis has passed off, and the brain disease has not yet proved fatal; the inflammation having subsided has left the optic nerves in the condition known as consecutive, or post-papillitic, or post-neuritic atrophy, the appearances of which are shown tolerably well in this diagram. In such cases there is generally gross defect of sight, and often complete or almost complete blindness.

Hemianopia, loss of the corresponding halves of the fields of vision is occasionally met with as a symptom of cerebral tumour. When this occurs, however, it is usually one of many symptoms; it may accompany hemiplegia, or hemianæsthesia, or both conditions, sometimes with aphasia also, and is due to destruction by the tumour of some portion of the fibres in the visual tract between the optic chiasma and the centres for vision. Hemianopia may co-exist with optic neuritis. As to the value of this symptom in diagnosis, and in localization of the tumour, I think my remarks

may be reserved until I come to consider it in connection with some other lesions of the brain in which it is more common. It is not a common symptom of cerebral tumour.

Oculo-motor paralysis in cases of brain tumour, by which I mean paralysis of some of the extra-ocular muscles—the recti or the oblique muscles, is now and then met with, but is not very common, except in cases of new growth situated at the base of the brain, and which produce this symptom by pressure on the trunks of the nerves (the third, fourth, or sixth) after they emerge from the cerebrum and before they leave the intra-cranial cavity. The kind of defect met with will of course depend upon the nerve or nerves involved, but to that we need not at present refer. This oculo-motor palsy may accompany other symptoms of pressure by the tumour; thus there may be hemianopia (as in a case I shall refer to in my next lecture), or atrophy of the optic nerve, and the tumour may at the same time interrupt the conductivity of one of the oculo-motor nerve trunks. The sixth is the nerve most commonly involved, probably because it has a much longer course to run over the base of the brain than either the third or fourth nerve. Tumours which are more centrally situated, namely, those which are not at the base of the brain, may destroy or interfere with the nuclei of the nerves, or with the nerve fibres in any part of their course between the nuclei and the point of emergence from the surface of the brain. There is, however, no difference in the symptoms thus produced by tumour from those produced by hæmorrhage, or softening following embolism or thrombosis. We may therefore defer consideration of them until we come to discuss the lesions produced by softening and cerebral hæmorrhage.

Abscess of the brain, one might imagine, a priori, would almost always be accompanied by optic neuritis, but as a matter of fact, although this symptom fairly often accompanies cerebral abscess, it does not occur in nearly such a large proportion of the cases as in those of cerebral tumour. The position and the acuteness of the abscess have a certain bearing on the occurrence of optic neuritis. As might naturally be supposed, an abscess situated near the base of the brain, and forming very rapidly, is more likely to cause optic neuritis than a chronic abscess situated in the frontal lobes, or anywhere near the vertex of the brain.

Now we come to meningitis, and under this

heading we will not include meningeal growths; they are more conveniently classified as intra-cranial tumours. All varieties of meningitis are frequently accompanied by optic neuritis. Here again, the position of the disease bears an important relation to the occurrence of optic neuritis, those forms of meningitis which are peculiarly prone to affect the base of the brain are very frequently productive of optic neuritis, whereas meningitis confined to the vertex may be unaccompanied by inflammation of the optic nerves; in fact, the position of the meningitis is of far greater importance than its kind as regards the production of optic neuritis. Hence it is, as I have said, that optic neuritis is more common in those varieties of inflammation, which specially, or exclusively, involve the basal meninges. These are the tubercular form, the syphilitic form, and the traumatic form, but especially the first and second.

In tubercular meningitis optic neuritis occurs with great frequency. In a series of fatal cases recorded by Garlick as occurring at the Children's Hospital, Great Ormond Street, and examined ophthalmoscopically during the whole course of the disease, optic neuritis occurred in 80 per cent.

In meningitis the optic neuritis is almost always symmetrical (affecting both optic nerves), although, as in intra-cranial tumours, it may vary in degree and in the date of the onset in the two eyes. The appearances of the optic discs are of no diagnostic value as regards the kind of meningitis present; the papillitis is exactly the same ophthalmoscopically, whether induced by tubercle at the base of the brain or by syphilitic or other disease, and it does not seem that the presence of optic neuritis has any decided prognostic significance in cases of meningitis.

In *tubercular meningitis*, another ocular lesion, namely, tubercle of the choroid, is sometimes met with. Tubercles in the choroid, although occurring in tubercular meningitis, seem to be less common in cases of meningeal disease solely, than in cases in which the patient is suffering from a general tuberculosis, with or without disease of the meninges. The drawing I now show is from a case of tubercle of the choroid occurring at one of the children's hospitals, in which there was general tuberculosis, affecting the lungs and most of the other organs, and there was also tubercular meningitis. Occasionally (as in the case I have just mentioned) the presence of tubercles in the

choroid has proved of considerable help in diagnosing between tuberculosis and obscure febrile conditions in children, usually cases of typhoid fever. Some cases of tuberculosis closely simulate typhoid fever in children; and in such instances the discovery of tubercles in the choroid would settle the diagnosis and probably have some bearing on the prognosis, the prognosis in general tuberculosis being infinitely worse than even in a bad case of typhoid fever.

In epidemic *cerebro-spinal meningitis* (which, fortunately, we do not often see), optic neuritis occurs in cases in which the patient survives longer than four or five days. As you are probably aware, this disease is generally rapidly fatal; even in those cases in which the disease does not kill in the first few days, the percentage in which optic neuritis is met with is not large. Schirmer, one of the few men who has written on this subject, mentions that he examined some 27 cases, and in only one of them was optic neuritis found. He, however, does not state (if I remember rightly) at what date in the illness the patients were examined ophthalmoscopically; therefore, perhaps, much reliance cannot be placed on these figures.

The last disease to which I shall refer to-day, is that known as *acute transverse spinal myelitis*, in which optic neuritis occurs not very infrequently. Cases of acute spinal myelitis are by no means common, but in those which have been recorded, optic neuritis has been met with in a considerable proportion. In such cases there may be no symptoms pointing definitely to intra-cranial disease, except this condition of optic neuritis. All the symptoms point to acute disease of the spinal cord, and in the reported cases attention has generally been drawn to the eyes by the presence of gross defect of sight, terminating rapidly in complete blindness. Optic neuritis is then found on ophthalmoscopic examination. These cases frequently prove rapidly fatal.*

Salicylic Acid in Ringworm.—Salicylic acid is highly recommended as an application to ringworm. It may be used as an ointment, but is much better as a saturated solution in collodion. One application is often all that is necessary to effect a cure, but more may be necessary. The pain caused is not usually severe.

(*Med. and Surg. Rep.*)

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* See "Trans. Ophthal. Soc.," vol. iv., 1884, page 232.

A CLINICAL LECTURE

ON

PERIMETRIC ABSCESS.

Delivered at the London Hospital by

G. ERNEST HERMAN, M.B. Lond., F.R.C.P.,

Senior Obstetric Physician to the Hospital, etc.

In many cases of perimetritis pus is present; but we do not apply the term perimetric abscess to cases of perimetritis in which we suspect a little pus in interspaces between adhesions. Whenever suppurative inflammation extends from one viscus to another, it is probable that a little pus is present between peritoneal adhesions. We mean by an abscess a cavity containing a considerable quantity of pus. Cullingworth, in fifty abdominal sections for recurrent peritonitis, found in thirty suppuration, either in tubes, ovaries, or peritoneum, intra-peritoneal abscess being present in two.

My own experience, and that of many others, is that the majority of inflammatory lumps in the pelvis get practically well; that is, they cease to cause symptoms, and get smaller or disappear. It is not the case that 60 per cent. of them persist and cause recurrent inflammation. The inference is, that a little pus in the pelvis may be absorbed. In hypopyon, the absorption of pus can be seen. In pyæmic suppuration of joints, pus is sometimes absorbed and the use of the joint recovered. Similar absorption of pus may take place in the pelvis; but when there is enough pus to be called an abscess, the patient does not get well until the pus has been discharged.

A large perimetric abscess is not usually a globular cavity, but one irregular in shape, having pouches in all directions. The condition begins as a number of little collections of pus and serum, in spaces formed by adhesions. These increase and coalesce, and thus a big abscess of irregular shape is formed. A globular suppurating cavity within the peritoneum is usually a suppurated cyst. But an abscess in Douglas's pouch may push up the bowels, and become globular in shape, and closely resemble an ovarian tumour. Barnes * relates such a case, in which the abscess was due to the bursting of an ovarian cyst.

Causes of Perimetric Abscess. The great majority of intra-peritoneal collections of pus are complica-

tions of purulent salpingitis.* Although in most cases of purulent salpingitis the fimbriated end of the tube is closed, yet in the early stage of salpingitis it is open, and pus may escape into the peritoneum, and thus an intra-peritoneal abscess be formed. I agree with Pozzi, that many cases described as pelvic abscess, cured by incision and drainage, were probably dilated and adherent suppurated tubes. The next most common cause of intra-peritoneal abscess is suppuration of the ovary; but this is commonly produced by extension of salpingitis. In a few cases suppuration in the ovary is produced by infection from the bowel. Disintegration or sloughing of uterine fibroids may produce suppuration in the peritoneum around.† Intra-peritoneal abscess may result by extension of inflammation from the bowel. Intra-peritoneal abscess is probably oftener due to bowel infection than the statistics of gynæcological surgeons show; for in many such cases the origin of the suppuration is clear, and the patient goes to a general surgeon and not to a gynæcologist. Intra-peritoneal abscess has been traced to a suppurating lumbar gland.‡ Lastly, as intra-peritoneal abscess is a disease from which patients often recover, in many cases the cause is not found out.

Diagnosis of Perimetric Abscess. In the beginning the symptoms of these cases are those of acute perimetritis, and it is not possible to say whether the inflammation is going to end in adhesions or suppuration. The patient remains ill for weeks, pain and fever do not subside, tenderness becomes more local, and when it has diminished enough for bimanual examination to be practicable, a tumour is found. An intra-peritoneal abscess can only be distinguished from an encysted intra-peritoneal collection of serum by the greater hectic fever and wasting which accompany suppuration. A collection of pus within the peritoneum big enough to distend the cavity containing it into a globular shape (which is rare) can only be distinguished from an ovarian tumour by its fixity, and its being accompanied by hectic.§ An ovarian tumour which has suppurated and become fixed by peritonitis around it, cannot be distinguished from an abscess in the peritoneal cavity unless the abscess be pointing.

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* "Diseases of Women," 1st edition, p. 363.

* See Cullingworth, "Birmingham Med. Review," 1893.
† See a case by the author, "Lancet," Dec. 8, 1894.
‡ Cullingworth, "Birmingham Med. Review," 1893.
§ See Barnes *loc. cit.* for a case illustrating this difficulty

Ovarian Suppuration without fever. Suppurated ovarian cysts do not always cause fever. Probably this is because the fever is due to a phlogogenic poison produced by microbes, readily taken in by the lymphatics in the peritoneum, but less easily absorbed by the wall of an ovarian cyst. A suppurated ovarian cyst without fever is fixed, commonly produces pain, and slow wasting; the nature of the tumour is generally doubtful until the abdomen has been opened.

When a small intra-peritoneal abscess has formed round a diseased tube or ovary it is not possible to diagnose the situation of the pus until the belly has been opened. The fact of suppuration is inferred from the persistence, in spite of treatment, of pelvic tumour, pain, and fever.

Remote Perimetritis. The fact that the inflammation is "*perimetric*" implies that adhesions have shut off the area of inflammation from the rest of the peritoneal cavity. In perimetritis there may be at one place simply adhesions, at another serous exudation, at another a collection of pus. A collection of pus may burst and empty itself while another collection of pus is getting larger. Thus inflammation in the pelvis may have subsided, while at another part of the peritoneal cavity suppuration is going on, which may thus appear to be independent of any pelvic cause, although it started from the pelvis and is really perimetric inflammation. This is called "remote perimetritis." For correct diagnosis in such cases a knowledge of the course of the illness is required.

Clinical course of Perimetric Abscess. When perimetritis has gone on to suppuration, fever and wasting continue until the abscess bursts or is opened, and the pus is discharged. This event proves that the swelling was an abscess; but the intra-peritoneal situation of the abscess in many cases remains a conjecture. This statement applies to large collections of pus. Small ones may be absorbed.

An encysted intra-peritoneal abscess may burst into the general peritoneal cavity, and set up fatal peritonitis; but this is rare. An intra-peritoneal abscess usually bursts into the bowel. It is easy to understand why: a large surface of bowel, with a comparatively thin wall, forms part of the wall of every perimetric abscess. I offer no estimate of the frequency with which intra-peritoneal abscesses discharge into the bowel, because in most such cases the diagnosis is a conjecture. When there is a tumour with hectic fever, and simultaneously the tumour disappears, fever ceases, and pus is passed by the bowel, the diagnosis is almost certain. But the abscess may burst high up so that the pus is mixed with fæcal matter and cannot be recognized; and then, although from subsidence of the tumour and improvement in symptoms you conjecture that an abscess has burst, yet certainty is not possible.

When a perimetric abscess has burst into the bowel, the course of the case depends on the cause of the suppuration. However, if the cause has ceased to exist, the abscess will close, and the patient get well. If there is a persistent cause, such, for instance, as a suppurated ovarian cyst, or an extra-uterine sac containing bones,* the abscess cavity will go on discharging indefinitely, and the patient may ultimately die from exhaustion, or from lardaceous disease.†

When an abscess bursts into the bowel, the pus escapes, but fæces, as a rule, do not get in. This is because (1) the abscess wall is usually under tension, and contracts when the pus has found vent; and (2) the opening is generally small, and, when the bowel contracts, the muscle in its wall temporarily closes the opening. Now and then fæcal matter does escape into an abscess cavity. I have known the pressure of an india-rubber tube, put into an abscess cavity to drain it, lead to perforation of bowel in contact with the tube, and escape of fæces into the abscess cavity.

It is often stated that perimetric abscesses may burst into the vagina, the bladder, or externally. A perimetric abscess may burst in more than one place. Duncan states that he has a preparation of a perimetric abscess opening into bladder and rectum.‡ But abscesses bursting elsewhere than into the bowel are more often in the cellular tissue than in the peritoneum; and in many of them it is not possible to say, until the belly has been opened, what the relations of the abscess are.

The pus of a perimetric abscess is sometimes mixed with blood, either because the abscess was a suppurated hæmatocele, or from hæmorrhage from the abscess wall. Often, but not always, the pus is

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* Suppuration from this cause, although it cannot clinically be distinguished from a perimetric abscess, yet is oftener under the peritoneum, in the cellular tissue, than in the peritoneal cavity.

† For an instance of the latter, see "St. Bartholomew's Hospital Rep.," vol. xvi., 1880, appendix p. 32, case 105.

‡ Perimetritis and Parametritis, p. 161.

very offensive, especially if the abscess is close to the large bowel. No inference as to diagnosis or prognosis can be drawn from the character of the pus.

Some abscesses seem to have no tendency to burst. Matthews Duncan states that he has opened abscesses which had been present for years, and showed no tendency to point. This peculiarity he believed to be one of perimetric abscess only : but he does not state upon what grounds he considered the abscesses he mentions to have been perimetric.

Treatment of Perimetric Abscess. The treatment (other than that common to all forms of perimetritis) is, first, to let out the pus. Sometimes this is enough. In other cases it is not: we must go further, and remove the cause of the abscess, which is generally a suppurated tube or ovary. The best way of attaining these ends depends upon the position of the abscess. It may be :

1. *In Douglas's pouch.* If there is a fixed (that is, adherent) swelling behind the uterus, which from the history and symptoms we take to be an abscess, matters cannot be made worse by opening it from the vagina, provided this be done with clean hands and instruments. Some do not approve this course, on the following grounds : (*a*) this measure does not inform us of the state of the tubes and ovaries ; (*b*) if these parts are diseased, they cannot be perfectly dealt with in this way; if, for instance, the abscess be caused by a suppurated ovarian cyst, it will go on discharging : and if there should be more than one cyst in the diseased ovary, the other cysts will go on growing. I think these objections not weighty, for the following reasons : (*a*) Some suppurated ovarian tumours can be cured by vaginal incision. Dermoid cysts, which are the ovarian tumours most liable to suppuration, are generally unilocular, and do not go on growing indefinitely. I have collected evidence* to show that when dermoid cysts are opened and drained by the vagina, the sinus shrinks and the discharge becomes so slight, that the patient experiences no further inconvenience. (*b*) I have cured abscesses that I believe were suppurated tubes, and so have others, by opening them through Douglas's pouch, although, for reasons explained above, it was impossible to be sure how the boundaries of the pus cavity were formed. (*c*) The removal of adherent suppurating tubes and ovaries is a difficult operation. The immediate risk to life, and

* "Obst. Trans." vol. xxvii.

the more remote risks, of an incomplete operation, so that the patient is left with a sinus discharging externally, and of a ventral hernia, are avoided if the abscess is attacked by the vagina. If the attempt to cure by vaginal incision fail, and a pelvic tumour persists, abdominal section can afterwards be done, and as the tumour was adherent before the vaginal incision was made, no difficulty will have been added to the case by that incision.

How to open it. Hands and instruments having been made surgically clean, put two fingers of the left hand into the vagina. Guided by them, cut with a pair of scissors for about an inch and a half from side to side through the mucous membrane of the posterior vaginal fornix, about halfway between the lowest part of the downward bulging tumour and the cervix uteri. If the tumour is an abscess, the mucous membrane will be its toughest covering. Having cut through this, try and push your fingers on into the abscess cavity. If the tissues are softened, as they often are, you will do this easily. If you cannot, cut carefully on with scissors, feeling as you go on the thickness and character of the tissues you are cutting through, and tearing your way onward with the fingers as soon as you can do so. When you have got one finger into the abscess cavity, enlarge the opening by tearing until you can get two fingers in. Explore the cavity ; feel if there be any solid contents, such as hair or bones, and if so, empty it. Lightly stuff the cavity with iodoform gauze. Free hæmorrhage from an opening in Douglas's pouch is rare, no large vessels being in this situation. If there should be more bleeding than is safe, stop it by putting a thick plug of iodoform gauze into the wound ; or tie a piece of string round the middle of a clean sponge and insert the sponge astride the opening. I think a free opening better than tapping with a needle, because by the latter the cavity can be neither explored nor emptied. Making the opening as much as possible by tearing with the fingers has the advantage that a tear bleeds less than a cut, and that softened tissues tear more easily than healthy ones, so that the wall of the pus collection is more likely to give way to your fingers than healthy tissue. Therefore by tearing you get free access to the pus collection with less risk of bleeding and of wounding what ought to be avoided, than if an opening of the same size were made with knife or scissors.

2. *Abscess above Douglas's pouch.* The lump may be behind and on one side of the uterus, but

higher up than Douglas's pouch. Cullingworth has pointed out that it is often high up when the inflammation results from puerperal infection, because while the puerperal uterus is large, the ovaries and tubes are above the pelvic brim ; hence, if inflammation attack the pelvic peritoneum by way of the Fallopian tubes before the uterus has sunk into the pelvis, the uterine appendages will get fixed in a higher position than that which they occupy in the unimpregnated state, and an abscess around them will be higher up also. When the abscess is thus situated, you cannot open it from the vagina without risk of wounding large vessels in the broad ligaments. Therefore, in such a case, as soon as it is clear that under expectant treatment the patient is getting worse instead of better, surgical treatment is indicated, and the best mode of treatment is by abdominal section.

3. *Abscess above the pelvis.* The abscess may be above the pelvis, apparently not connected with the uterus. The cases which call for interference are those in which the pus lies under the abdominal wall. In these cases we have a fixed tumour, adherent to the abdominal wall, with hectic fever and wasting. It is generally impossible to say whether such a tumour is an abscess or a new growth with inflammation in it and around it. If the patient is getting worse the proper course is to explore such a tumour by cutting through the abdominal wall over it. After the swelling has been opened and found to be an abscess, it is often impossible to be sure that it is intra-peritoneal, for the adhesions which bound it shut off the peritoneal cavity and cause the interior wall of the abscess to feel like a smooth continuous membrane. Although the shape internally of such an abscess is anfractuous, yet on abdominal palpation before being opened it may feel globular. Having opened it, drain it by lightly stuffing with iodoform gauze.

Hæmorrhagic Diarrhœa.—The *Medical and Surgical Reporter* recommends the following :

R. Pulv. alum.
Catechu   ...   āā   gr.v
M. Make four pills. Six pills per day.

R. Alum.
Catechu   ...   āā   gr.ij
Crude opium ...   ...   gr.¼
Syrup of red roses q.s.

M. Make one pill. Twelve pills per day.

CLINICAL
DEMONSTRATION OF CASES.

Given at the Monthly Meeting of the North-West London Clinical Society, held at the North-West London Hospital on Wednesday, March 20, 1895.

DR. HARRY CAMPBELL in the Chair.

Tuberculide of the Face.

DR. ABRAHAM showed a case of what he considered to be tuberculide of the face, in a woman 50 years of age. Soon after he first saw the patient, about three years ago, he brought the case before the Medical Society of London as one of syphilis of the face. It was then much more swollen than now, and there was a certain amount of ulceration with distinct serpiginous margins. The suspicion of syphilis was strengthened by the fact that the woman had had four miscarriages. There was, however, no history of the primary disease, though such is seldom forthcoming in such cases. He had given her iodide of potassium with marked improvement within a month. He continued to treat with iodide of potassium and some mercurial application, but there was no further improvement, and he ceased the treatment as there were signs of iodism, whether the dose was large or small. Since that time various forms of treatment had been adopted, but no improvement set in until six months ago. In an earlier stage, the face was so much swollen, that the question of a new growth entered his mind. For the last six months she had been taking extract of thyroid gland with marked improvement. External applications were discontinued owing to the irritability of the skin.

DR. STOWERS had no hesitation in regarding the case as one of lupus, an intermediate form having characters common both to lupus erythematosus and lupus vulgaris. This view was supported by the duration of the affection and its diffusion, although characteristic tubercles did not exist. It was a sufficiently unusual manifestation of the affection to give grounds for an opinion that it was tuberculous, but he believed it to be more closely related to lupus vulgaris than to lupus erythematosus.

DR. SIBLEY regarded the case as lupus erythematosus.

MR. JACKSON CLARKE asked whether the possibility of it being mycosis fungoides had occurred to Dr. Abraham. A peculiarity of this was that some of the small tumours would disappear and others come in their place. He thought more experience of the value of thyroid treatment in skin diseases was required before a definite opinion could be formed.

MR. MAYO COLLIER agreed that it was an unusual example of lupus. He found many patches on the scalp, which led him to believe that it was lupus erythematosus. It looked like a very extensive case of irritation, with local œdema around it. In his opinion lupus vulgaris might be excluded.

DR. CAMPBELL asked if Dr. Abraham would give his experience of the action of thyroid extract in cases of lupus.

DR. ABRAHAM, in reply, said he did not think it was a case of mycosis fungoides, though he had not made a microscopical examination. The scalp affection was recent. No one who saw the case at the Medical Society's meeting thought it was a form of lupus. He had had many cases of abnormal lupus, which he preferred to call tuberculoid. On the question of thyroid treatment, in several of his cases there had been very much improvement. Of one case he had showed photographs at a meeting of the Hunterian Society. Since that, however, the case had got worse. He had a similar case at the present time. He was inclined to believe that the thyroid so altered the nutrition of the tissue, as to assist in the removal of the putrefaction, and

probably at the same time assist in the formation of new tissue. He did not see how the thyroid was to cause the absolute disappearance of the lupus, having regard to the cause of the disease. Still, the benefit resulting from the extract was remarkable.

Tubercular Disease of the Nose and Palate.

DR. WILLIAM HILL showed for diagnosis a boy of about 15, with disease of the nose and palate. Although he brought it forward for diagnosis, he had not much doubt of its nature himself. He had had many somewhat similar cases (which he believed were usually called syphilis of the nose) in which perforation of the palate took place, and in which the ulcerations were pronounced. His opinion was that many of them were tuberculous. In this case the giant cells were present. He brought forward the case to appeal to members. Syphilis could practically be excluded. The glands in the neck were very large, and some of them on the point of suppurating. He had the ordinary tubercular lip, and there were granulations from the septum to various parts of the nose. On inverting the lip, the gum and palate were found ulcerated, and further back the palate was perforated. This latter point might lead many of them to regard the disease as syphilitic, but he was almost certain it was tubercular, and he would describe it as tubercular lupus. It was supposed to be of two months duration, but he thought it must be much older. The patient had been treated with iodide of potassium.

MR. MAYO COLLIER thought it was a rare instance of a combination of strumous glands of the neck and strumous septum of the nose, and he was inclined to agree with Dr. Hill's diagnosis. In the old days it would be called scrofula. The perforation of the palate was probably congenital, or at any rate of many years standing. The cicatricial tissues showed no evidence of recent action, nor in the nose, as shown by the granulation thickening in the septum. He did not agree that there was lupus of the gums, because very frequently, in persons of low vitality who did not keep their teeth clean, there were the same appearances. He considered it simply ulceration due to the bacteria which congregated there. The patient would get well more or less rapidly under cod-liver oil, iodide of iron, and good feeding, and simple washing of the nose.

DR. CAMPBELL, in expressing agreement with Mr. Collier's opinion, thought the condition of the teeth and gums was independent of any tubercular process. A most interesting point in connection with tubercle was the way in which it picked out certain parts of the body. Doubtless pathologists were able to account for this. As to treatment, mercury was an exceedingly good drug for struma. He had a pet theory on tubercular disease, namely, that it was due to a double process, starvation of the tissues and poisoning of the tissues secondary to chronic catarrh of the bowel. It was quite a common occurrence for poor children to come to the hospital with large stomachs, prominent veins, and foul-smelling motions, which were abnormally coloured and containing slime, showing all the symptoms of chronic enteritis. In all strumous cases of the joints there was chronic poisoning by ptomaïnes, which reduced the vitality of the parts of the body, and rendered them more prone to tubercle. In such cases he believed it very important to pay attention to the condition of the intestines, and it was an ailment in which calomel was of great benefit.

DR. HILL, in reply, said the patient had been taking grey powder for three weeks. He thanked the speakers for the suggestions which they made, and would bear them in mind, Those who had looked carefully at the patient would see that the ulceration went right up to the fornix, and almost into the nasal cavity. Further, the fangs of the teeth were becoming exposed. He was applying lactic acid locally, and later on he intended having the nose surgically treated.

Dupuytren's Contraction of the Fingers.

MR. JACKSON CLARKE brought forward a case of Dupuy-

tren's contraction of the fingers of both hands, the patient being a man aged 71. There had been pain in both shoulders for twelve months, but the deformity of the hands had only been present for three months, and had been accompanied by pain in the palms and by redness around, also pain in the metacarpo-phalangeal joints. The latter he took as an indication that the deformity was of a gouty nature, although there had never been inflammation in the great toe joints. In some dissections of hands affected by this deformity, urate of soda had been found in the nodular thickenings of the palmar fascia. Observations recently published by Dr. Berkhart had fully demonstrated the fact that the deposition of urate was only a secondary event in gout, and was preceded by the formation of inflammatory tissue, or even of foci of new growth. Mr. Clarke had been able to confirm the observations of Dr. Berkhart, who had kindly lent some microscopic slides which were demonstrated to the meeting, and which showed the crystals deposited in necrotic foci, surrounded by a zone of endothelioid and giant cells, with some formation of new capillaries. Mr. Clarke thought cicatrisation, following on such pathological changes, fully accounted for the deformities. As to treatment, operative interference was out of the question so long as inflammatory changes were present. This patient had greatly improved on salicylate of soda and local rubbing. When there were no signs of active gout in any part of the body, and when the hands had been free from inflammatory symptoms for at least twelve months, operative measures might be contemplated. If there were but few adhesions of the thickened process of fascia to the skin, multiple subcutaneous section was indicated, but if the skin was extensively adherent to the process of fascia, excision of the latter was indicated. Either operation could be done under cocaine. For subcutaneous injection, he recommended a 1 per cent. solution in a 1½ per cent. solution of boric acid. By testing this upon himself, Mr. Clarke had found that it caused no pain at the moment of injection, and in one minute produced anæsthesia, which lasted fifteen minutes. He felt sure that subcutaneous solutions of cocaine were frequently used far too strong. In anæsthetising the skin, it was important to inject the solution into the corium. When this was done, a white wheal marked the extent to which the liquid had diffused. The only danger was in injecting a considerable quantity of the solution into a vein. This could be avoided by only injecting a small quantity at any one spot, or, in other words, by pushing the point of the needle gradually onwards as the injection was being made. The solution, the skin, the needle, and the syringe should be sterilised before the operation.

Osteotomy of the Femur.

MR. JACKSON CLARKE showed a case of osteotomy of the femur for genu valgum. It was then six months after the operation. The patient was a rather stout girl, aged 12. A photograph, taken before the operation, showed a marked genu valgum of the right side and a slight inward curving of the left tibia. In the photograph, the patient was leaning on a chair for support. She had assumed this position naturally, in consequence of the relative weakness of the right lower limb. Before the operation there was scoliosis, with the dorsal convexity to the left. At the present time the patient had been walking about for over three months, and the right leg was now stronger than the left, and the genu valgum was entirely corrected. There was now some tendency to scoliosis in the opposite direction to that observed at first. In order to prevent permanent changes in the spinal column, and to improve the gait it would be necessary to divide the left tibia and fibula, and to straighten the leg. The bones were too hard to admit of any improvement by means of splints.

MR. COLLIER did not think the limb would be straightened in the way described, and suggested that Mr. Clarke should leave well alone, and not proceed further, as the deformity would be hidden by the dress.

MR. CLARKE was afraid that if it were not corrected the deformity would become very marked, and cause spinal trouble later on.

MR. GORDON BRODIE agreed with Mr. Clarke, and thought the course he suggested a wise one.

A Case of Friedreich's Disease.

DR. GUTHRIE showed an early case of Friedreich's disease, in a boy 10 years of age. His parents were both alive and healthy, and, so far as he knew, showed no signs of nervous disease, nor did other members of their families. There were three other children of the marriage who were perfectly healthy. The patient was born under normal conditions. At 12 months of age he had rickets, and became bandy-legged. He walked at the usual time, but was afterwards unsteady on his legs. As the unsteadiness increased, the patient was brought to Dr. Guthrie. He had difficulty in standing and the tendons were in continuous motion to preserve equilibrium. When he shut his eyes he would fall, if not supported. He walked on a wide base as if alcoholic. He also had other signs of ataxy. There was difficulty in bringing the points of the fingers together. He could write fairly well ; he held the pencil tightly, but was apt to drop it, and sometimes it would fly off at a tangent. The letters were well shaped, but jagged. As to the eyes, the pupils were equal, and reacted to light perfectly ; the discs were normal. He had no deformities. The knee-jerks were entirely absent, and had been as long as Dr. Guthrie had known him. His sensation was perfect to touch, heat, pain, and cold. He had called it a case of Friedreich's disease because the symptoms were more of locomotor ataxy rather than of any other form of disease. It was well known that there were isolated cases in a family, but some of the other children might become similarly affected hereafter. The tremors suggested that it was a case of disseminated sclerosis, but he was too young for that, the knee-jerks were absent, and the tremors were not of the kind found in this affection. It might be confounded with chronic diphtheritic paralysis, but here there was no history of diphtheria, and such cases were extremely rare.

DR. CAMPBELL agreed with Dr. Guthrie, and asked what prognosis he gave in the matter.

DR. GUTHRIE replied that the prognosis was bad eventually, but in this instance it was not now unfavourable, as such cases lingered on indefinitely. Cases of the kind might live to a considerable age, and they might die of some intercurrent affection. In very severe cases they went from bad to worse, and had deformities of the limbs, curvature of the spine, and inability to walk. Some cases would last for a number of years, appearing to get neither better nor worse.

Hallux Rigidus.

MR. COLLIER showed a case of hallux rigidus. He said it was a disease on the pathology and treatment of which the large number of text-books would give very little instruction. His first case was ten years ago, when he applied the treatment recommended in the standard works, but it failed. The tendons were divided, and splints were used, but the result was only disappointment. He therefore cut down to the joint, and found undoubted caries between the head of the metatarsal bone and the sesamoid bones. Seeing that it was pressure caries he determined to remove the head of the bone and scrape the remaining portion of cartilage, and close the joint immediately. Healing took place in ten days, and the girl could walk without disability. She could stand on tip-toe, and the only trace of an operation was that the toes were somewhat shorter. The disease was nearly always associated with a degree of flat foot. Such patients showed defective nutrition in the limb, which was usually cold and bluish. Mr. Collier pointed out the distinction between hallux rigidus and hallux flexus. He had operated on fourteen cases, and every one showed pressure caries.

MR. JACKSON CLARKE was of opinion that nothing short of operation would give the slightest benefit.

Graves' Disease

DR. CAMPBELL showed a case of Graves' disease in a young man of 19. He presented all the classical symptoms—exophthalmos, large thyroid, palpitation, and tremor. The thyroid was so large that it pressed on the trachea and interfered with breathing. He came for more treatment for asthma. Tremor was believed to be one of the classical signs of Graves' disease. His own opinion was that the tremor was connected with the palpitation. He had observed some hundreds of cases of palpitation, and tremor was present in all of them. He was of opinion that tremor from which women frequently suffer was really paralysis—slackening of the individual contractions, which were like tetanus. He was giving valeriante of zinc. The patient was considerably better now. Dr. Campbell considered that minor cases of Graves' disease were very common. In reply to a question from Dr. Herschell, Dr. Campbell said he had not looked to see whether the stomach was dilated, nor tested the electrical resistance of the body.

Lupus Vulgaris.

DR. STOWERS showed a boy of 15, suffering from lupus vulgaris on the right buttock. The patient had no disease elsewhere. It was painless, and had existed for eight or nine years.

MR. JACKSON CLARKE confirmed the above opinions, and felt certain that if injected under the skin of a guinea-pig, the animal would succumb to tuberculosis in fifteen days.

Two Cases of Granular Kidney.

DR. CAMPBELL exhibited two cases of granular kidney— a male and female, both about 50 years of age. Both patients came to the hospital suffering from influenza. They had albuminuric retinitis, thickening of the arteries, and hypertrophied hearts. It was curious that a sufferer from granular kidney might hunt on Monday, have albuminuria on Wednesday, and die on Saturday. The two chief causes were an attack of inflammation, and, secondly, a giving way of the heart. In such cases tracts of perfectly normal tissue would be found, and he believed it was these tissues which kept the patients going. If the heart suddenly gave way the circulation became weakened, and was no longer able to supply these limited tracts of tissue with sufficient blood, consequently the patient collapsed. From that he concluded that the giving way of the heart was a very common cause of granular kidney. There was accentuation of the second sound of the heart (aortic).

Prurigo.

DR. STOWERS brought forward a case of prurigo (hebra) in a boy 15 years of age. He said this disease was looked upon as rare in this country, much rarer than it really is. Cases of the disease in infancy become aggravated into ecthymatous and eczematous conditions, so that the original disorder was masked. The patient before them had an intensely pruriginous cutaneous disease for eight years, and the skin was markedly pigmented. He had a few of the original papules characteristic of prurigo superadded. There was much evidence of severe irritation and scratching. As a result of the severity and chronicity of the disease, he had well marked prurigo bubos. The enlarged glands were very large and hard, but had never attempted to suppurate. The prognosis was grave always. Our present knowledge only justified us in regarding treatment as palliative. Immediately the patient came into the hospital, he began to improve, but on leaving it to return to less hygienic conditions, aggravation followed.

DR. STOWERS also showed two cases of warts, involving the face and hands, and described their nature and characteristics. One patient included a well-marked example of the planar variety.

THERAPEUTICAL NOTES AND FORMULÆ.

Treatment of Acute Bronchitis in Infants.
—Dr. Kerbey lays down the following indications:
—Changing of rooms, or good air temperature at
72° F.; warm dry clothing; keep the child in
crib, changing the position frequently. If bottle-
fed, give partially digested or easily digested foods,
daily evening sponge or bath in salt water; if seen
at the onset, three to five drops of castor oil every
two hours for two or three days, followed by anti-
mony and ipecac. in small doses. If condition of
heart require it, give whisky, 10 to 30 drops, or
strophanthus, one to two drops, every hour or two;
for restlessness, small doses of bromide and chloral,
or Dover's powder, ⅛ to ¼ grain. Mustard, or
steam spray, as counter-irritants.

<div align="right">(Archives of Pediatrics).</div>

The Treatment of Vomiting in Children.—
The *Journal de Clinique et de Therapeutique*
publishes the following directions and formulæ to
be used in the treatment of vomiting in children.
Very young children should be made to swallow
small pieces of ice before nursing. Milk diluted
with a little Vals or d'Alet water should also be
given. Before the child is nursed, 3¾ grains of
bismuth subnitrate should be put on its tongue.
The diet should be restricted, the milk sterilized,
and the time of nursing properly regulated. For
older children, iced drinks, ice, and effervescent
waters are recommended. A teaspoonful, each,
of the following mixtures is to be taken, be-
ginning with the first:—

1. Potassium bicarbonate ... gr.xxx
   Syrup ... ... gr.ccxxv
   Water ... ... ʒiss
2. Citric acid ... ... gr.xxx
   Syrup of citric acid ... ... gr.ccxxv
   Water ... ... ʒiss

Fonssagrives recommends the following:—

Essence of cajuput ... ad gtt.vj-xij
Sugar ... ... gr.xxx

When this is thoroughly mixed, add an ounce of
syrup of tolu and three ounces of Melissa water.
From a teaspoonful to a tablespoonful is to be
taken every hour. Huchard prescribes 75 grains
of tincture of iodine and 225 grains of saturated
chloroform water, of which from two to six drops
are to be taken in a little sweetened water.

For nervous children, over 12 years old, Ewald
prescribes—

Cherry laurel water ... ... ʒvj
Tincture of belladonna ... ... gr.lxxv
Cocaine hydrochloride ... ... gr.ivss
Morphine hydrochloride ... ... gr.iij

From five to ten drops are to be taken every
hour or two.

The following formula is recommended by
Guibourt:—

Syrup of lemon ... ... gr.ccc
Lemon juice and
    orange flower water, each... gr.ccxxv
Linden water ... ... ʒij
Sydenham's laudanum ... ... gr.ix
Sulphuric ether ... ... gr.xv
Potassium bicarbonate ... ... gr.xxx

The bottle should be corked immediately, and
from a quarter to a third of the mixture is to be
taken at once. Le Bariller advises the use of the
ether spray over the epigastrium; also blisters or
the actual cautery over the same part.

Borax as an Aid to the Digestion of Milk.
—M. Germaine See has recently announced the
clinical fact that borax used internally is a valuable
aid to the digestion of milk. He discards the use
of carminatives, charcoal, and other intestinal anti-
septics, claiming that they injure the mucous
membrane of the intestines. He employs laxatives
—hydrastis canadensis, castor oil, and olive oil in
large doses, or oil enemata. Prof. See holds that
in many cases of indigestion the stomach is erro-
neously treated when the real cause of the disease
is the intestines, which are often the seat of mem-
branous enteritis resulting from constipation, and
giving rise to glairy, mucilaginous, cylindrical
masses of mucus, with pain and swelling over the
region of the colon. These symptoms easily dis-
tinguished the cases referred to from ordinary
constipation, in which there may be easily seen
masses of filamentous or vermicelli-like mucus.

<div align="right">(Med. and Surg. Rep.)</div>

Legumine as a Substitute for Milk Diet.—
Bovet relates four cases of gastric disorders where
milk diet was not well borne, and legumine in
high doses as 16 drachms a day succeeded in
adults. Legumine is not only a medicament, but
a food, owing to the phosphated and albuminoid
elements it contains.—(*Therap. Gazette.*)

Hæmatemesis from Gastric Ulcer.—Enjoin absolute rest in bed, forbidding motion or speech. Apply an ice-bag over the epigastrium. Order iced drinks, ice to suck, and every two hours a cachet containing

Sodium bicarbonate	...	gr.7¾
Ratanhia powder	...	gr.1¾
Opium powder	...	gr.⅙

Give a hypodermatic injection, every half-hour, of 15½ minims of the following solution:

Ergotin	... ...	gr.31
Glycerin	... ...	f ʒi¼
Cherry-laurel water	...	f ʒi¼

As food, a glass of iced milk every two hours, and an injection of yolks of two eggs, 2½ drachms of dry peptones, and 9½ fl. oz. of milk. Between the attacks have the patient observe a strict milk diet, taking a glass of fresh milk every two hours, with a teaspoonful of dry peptones if the pain is not too severe. Follow the milk with bicarbonate of soda, giving a little in water after each glass of milk. If pain is very severe, prescribe the following:

Hydrochl. of morphine		gr.⅙
Hydrochlor. of cocaine		gr.7¾
Cherry-laurel water	...	f ʒ2½
Julep ...	... ...	f ʒ3¼

A tablespoonful three times a day.

Continue the treatment until pain has completely ceased, when food may be gradually resumed, preparations of milk, eggs, and meat powders being first allowed, milk with Vichy water being the only drink.—(*Tribune Médicale.*)

Guaiacol Carbonate.—Used in 160 cases of phthisis by author, 31 to 46 grains daily in two doses, increasing gradually to 1½ drachms when indicated. Preferable to creasote or pure guaiacol because it does not disturb digestion, is taken into the blood in small quantity only, and exerts a milder influence. Appetite and assimilation of food improved. Valueless in acute miliary tuberculosis.—(HÖLSCHER, *Berliner Klin. Woch.*)

Painful Dentition.—The *New York Polyclinic* gives the following:—

℞ Muriate of cocaine ...	...	gr.iss
Tr. of conium ... ⎱	āā	ʒij
Syrup ... ... ⎰		

M. Rub on the gums several times daily.

Chloral in Ear-ache.—Brodnax has the following remarks about the use of chloral in earache in his paper contributed to the *Polyclinic*:

℞ Camphor	... ...	gr.x
Chloral	... ...	gr.x
Carbolic acid ...	...	gr.x
Castor oil	... ...	ʒss

Drop into the ear warm.

Fill the ear full, apply a piece of cotton wetted in warm water to fill the external ear, then a cloth wrung out in hot water as warm as can be borne.

REVIEW.

Indigestion. By GEORGE HERSCHELL, M.D. (Ballière, Tindall and Cox.)

TAKING this book all round we feel that we must congratulate Dr. Herschell on the ability which he has shown in condensing and arranging the voluminous literature, chiefly Continental, which has appeared on the subject of dyspepsia. Judging from a perusal of the many various manipulations which the material withdrawn from the stomach has to undergo, we can only conclude that a whole laboratory and unlimited leisure must be required to make one a competent authority on normal digestion; and, alas! at the end of our investigations it seems to be invariably found that some other authority has arrived at precisely the opposite conclusion. We would not for a moment, however, be suspected of sneering at the work that has been done: we only wish to draw attention to the enormous difficulty of the subject, while expressing a hope that at some not far distant day we may get a really authoritative statement on it. Meantime, those who read Dr. Herschell's book will find many useful hints, especially on treatment. He prints in italics a rule which seems too good not to deserve quotation: "Never forbid any article of diet unless there is a distinct reason based upon the pathological aspect of the case why it should not be taken." Whether all patients are as docile as Dr. Herschell's seem to be in allowing intubation of the stomach we should doubt, but otherwise the advice given is reasonable and to the point, and we think the book is one which may be read with pleasure and profit, even by men of little leisure.

THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON THE

ARTIFICIAL FEEDING of INFANTS.

Delivered at St. Bartholomew's Hospital, Feb. 11, 1895,

BY

WALTER S. A. GRIFFITH, M.D., F.R.C.P.,

Assistant Obstetric Physician to the Hospital.

GENTLEMEN,—My subject to-day is the artificial feeding of infants. Infants are required to be fed artificially from various causes, such, for instance, as the mother's death, or her inability, for some reason, to feed her offspring herself. In such circumstances, how is the baby to be fed? A little experience of these cases will impress upon you the necessity of having definite principles to act upon, and of giving precise and minute directions to nurses, who in this respect are usually very imperfectly trained; for however able and trustworthy a monthly nurse may be in points connected with the nursing of the mother, it is an exceptional thing to find one equally capable as regards the management, and especially the feeding, of the infant. A nurse who thoroughly understands the management of the baby as well as of the mother, one on whom you can rely, both for her judgment and in the carrying out of all the details of baby feeding, will be found a most valuable and not common acquisition to you in your practice.

We may conveniently put the matter before us into two propositions:

(*a*) In order to feed a baby successfully by hand, as it is called, it is essential to imitate the natural process as nearly as possible in the following particulars:

1. In the composition of the food.
2. In the quantity to be given.
3. In the temperature of the food (a thing which is often forgotten).
4. In the frequency and rate at which it is given (what I may call the administration, for want of a better term). As much time should be occupied in taking food from the bottle as from the breast.

5. Finally, the food should be quite free from contamination.

These five points we shall have to consider in detail.

(*b*) Ailments of young babies born healthy are mostly due to errors in feeding. If you find a young infant ailing, being born healthy and free from any grave injury, the most common cause is that there is something wrong with the food, whether nursed by the mother or artificially. When we consider what a large proportion of deaths occur in the first year, you will see how important this matter of food and feeding is. Of course some ailments at this period of life may be due to exposure to cold. Therefore, in the case of such an ailing infant your first duty is to go minutely through the points I have mentioned in regard to its food—quality, quantity, rate of feeding, temperature, freedom from contamination, and find which of them is in fault.

To properly understand the artificial feeding of infants, it is essential that you should understand the natural process. This involves details by which you will be able to distinguish the experienced and capable from the careless and ignorant nurse. It must be remembered that our duties as doctors do not end with the delivery of the child; we require to see how the baby as well as the mother progresses. If you find the baby is not doing very well, tell the nurse to put it to the breast. Admittedly this is a very simple matter, but watch how she proceeds to do it. A careless, ill-instructed nurse merely gives the mother the baby and tells her to feed it, while the careful nurse will take a great deal of trouble to teach the young mother. If the baby is to be put to the left breast first, the nurse should turn the mother over to the left side, then lay the child on the mother's left arm in such a position that the mother can drop the nipple into the baby's mouth without the baby having to make any effort to take it. Watch a hungry baby struggling to get hold of a nipple; it knows perfectly well that matters are not right. It also knows a full from an empty breast.

A careful nurse will see that the infant is put to the breasts alternately, and that it is only put to one at a time. If the secretion is natural, the milk from one breast is sufficient for one meal. Another very important detail is that the mother should put the baby to the breast at regular intervals. These intervals, of course, vary according to the time after delivery. When should the baby be first put to the breast? The answer to this question will indicate when we should first artificially feed the baby. We all know the usual points to be observed after delivery. The mother has some food, hot beef-tea, hot milk, or whatever of a light character she fancies, the room is darkened, and the patient sleeps for two or three hours. On awakening she has more food, given by the nurse, and after this second food is the time for the baby to be first put to the breast. That may seem a very small matter, yet it is an important one.

You will find a good deal of difference in babies immediately after birth. One may hardly wake up, except when it is soiled, while another will behave quite differently, and will show that it is hungry from the first, and it must be fed occasionally, but in ordinary circumstances two or three times in the first 24 hours is sufficient. After the first 24 hours, the baby will want to be put to the breast a little more frequently. As soon as the milk begins to be secreted in a larger quantity, that is, in the fourth period of 24 hours (sometimes in the third period), when the breasts become distinctly full, let the infant be put to the nipple every two hours regularly for the whole of the first month. As the baby gets older, the interval should be gradually lengthened to every 2½ hours. It is very important that feeding should not take place between those times, the infant's stomach requires intervals of rest, just as the stomach of an adult does. If the conditions are natural, the mother should not suffer pain when the baby is put to the breast. The moment you see the mother's face screwing up when the child is put to the breast, you may suspect that there is something wrong with the nipple. I will not go into that now, except to say that lactation should not produce any pain in the nipple, but there is usually a very marked, and often painful but beneficial reflex contraction of the womb set up by putting the infant to the breast. In some women these contractions are comparatively painless, but they are always beneficial. They squeeze out any *débris* and clots

which may have accumulated, and are very important.

Again, there should be no rise of temperature during lactation. If there is one mistake more common than another on this subject it is in supposing that the influx of milk (which of course is an influx of blood leading to the formation of milk), is physiologically attended by a rise of temperature. Consequently, when the breasts are beginning to get distended, and the temperature of the patient, previously normal, goes up to 102°, one hears people say, "It is nothing, it is only the milk coming in." You may be sure that the cause of such rise is not physiological, but pathological, and the nature of such cause must be ascertained. It may be your only favourable chance of dealing with a serious complication in an early stage.

Should anything be done to the mother's nipple after the baby has been put to the breast? It is essential during the first weeks, and is important all through lactation, that the nipple should be washed and thoroughly dried. A soft handkerchief will do for the purpose; then dust the nipple well with a little powdered gum or boracic acid. A woman who has borne several children knows when she has been well nursed, and it is these small details which help to distinguish the efficient from the inefficient nurse.

Those are the main points connected with natural lactation as regards the mother.

With regard to the infant, in the first place you should watch the baby suckling. A little practice will enable you to ascertain by the sound the baby makes in swallowing that it is obtaining the milk, and the appearance of quiet satisfaction is very different from the fretfulness of unsatisfied hunger. When the infant cannot get the milk there is no sound of swallowing, and when the child is simply pulling at the nipple without result it is damaging the epithelium, and a sore nipple will be very liable to occur. When the milk comes freely it pours into the child's mouth, and is taken without any effort on its part, it has got all it can do to swallow it. A baby fed naturally should sleep through the whole of the two hours' interval. A good nurse will, from the first, try to train the baby not to want the breast between certain hours at night. The breast should be taken at 11 o'clock or midnight, and not again until about 6 o'clock in the morning, or even 7. The mother needs six hours continuous sleep. But you must not suppose every baby will sleep for six hours at night; but

a good nurse will teach it, and at the end of the month there will be considerable improvement in this matter, and the child will only wake if it is wet or uncomfortable. Babies which lie asleep and do not cry are said to be doing well, but you have to be on your guard against what I may call the fraudulent nurse—the dishonest nurse. You will find that some nurses—who have been badly trained—are often in the habit of giving babies soothing powders. They know that if the doctor comes and frequently finds the baby crying there will be some dissatisfaction with the nurse. To prevent that they give the baby somebody's soothing powders, which contain one or two grains of bromide, and sometimes drugs more harmful than bromide. The sleep of the child under such circumstances appears to be perfectly natural. There is another drug used for the same purpose—namely, brandy; a little in milk. Five drops will often send a child to sleep for hours.

Now as regards the infant's motions. Meconium alone is passed for the first two or three days; during the third day it is seen to be mixed with digested milk, and soon after should be entirely replaced by it. What are the characters of a healthy infant's motions? These you must study before you can hope to distinguish the deviations from health. One of the most important indications of perfect or imperfect digestion is obtained by observing these points.

As a rule, a child begins to pass meconium within a few hours of its birth. In the course of the third day the excreta begins to assume a bright yellow colour, and by the end of the fourth day the whole of the meconium has been expelled. The motion is of a thick, almost viscid consistence, usually described by the nurse as being like thick custard. It should be free from solid masses, whether large or in flakes. You will recognize the importance of this when we come to the tests of the success or otherwise of artificial feeding. The motions should be passed without flatulence, without pain, and should be passed two or three times in twenty-four hours. When the baby is being brought up on artificial food, do not ask the nurse in general terms how the infant is doing; she will naturally answer in general terms, that it is doing pretty well. But ask particular questions: "How often have this baby's bowels acted during the last twenty-four hours?" Perhaps the answer will be "Not at all." Such a state of things is absolutely fatal to the welfare of a baby. On the other hand,

they may have moved every hour, which denotes a more dangerous condition of things. You should see the motion; it may be green or of a pale chalky consistence, with cheesy, hard masses.

The most important and certain test of the progress—satisfactory or otherwise—of the baby is obtained by ascertaining its weight. The man who attends midwifery cases and omits to have the baby weighed within the first twenty-four hours is as unreliable a practitioner as the man who waits for fever to develop before taking the mother's temperature. I believe it is a very common thing to omit this weighing. I must tell you, however, that in getting this done you will often have some difficulty to surmount. There is a most extraordinary prejudice, even among educated people, against doing it. It is regarded as being particularly unlucky, and serves to show what an amount of ignorance still exists. But, especially if the baby is to be brought up by hand, is it absolutely essential that it should be weighed exactly, otherwise you have no definite data to go upon. A highly experienced nurse can tell without weighing whether the child is gaining or losing weight; but this is not sufficiently accurate, we must know the exact amount of gain or loss.

How should a baby's weight vary during the first month? Though not invariably the case, it is common for the child to lose weight for the first three, four, or five days. This may be explained by the fact that the child is steadily losing its meconium, while the amount of food taken is very small—not sufficient to compensate for this loss. But at the end of the first week it should have made up its weight. (At Queen Charlotte's I have every baby weighed on the eighth and again on the fourteenth days, the latter being the day on which they leave the hospital.) Then from the end of the first week onwards it should gain an ounce a day. A baby which gains half an ounce a day is doing fairly well, but a child doing thoroughly well gains double that amount or more. You will be able to appreciate what an enormous gain one ounce a day means ultimately. This should continue until the fourth month, after which half an ounce *per diem* gain is satisfactory. You will occasionally find babies gain nearly a pound in a week, which of course is highly satisfactory. Now, supposing a baby which is being brought up by hand has lost eight ounces by the eighth day, and at the end of the next week it has only made up two ounces of that, being six ounces below the

weight at birth, you have definite information to go upon. You may say the baby does not look very bad, but all uncertainty as to its progress is banished by adopting the course I have mentioned, and you may be quite sure there is something wrong with the feeding.

(*To be continued.*)

TWO LECTURES
ON
OCULAR SYMPTOMS IN DISEASES
OF THE
BRAIN AND SPINAL CORD.

Delivered at St. Thomas's Hospital, March 1 and 11, 1895,

By J. B. LAWFORD, F.R.C.S.,

Ophthalmic Surgeon to the Hospital, and Lecturer on Ophthalmology in its Medical School.

LECTURE II.

GENTLEMEN,—To-day, we begin by a consideration of the ocular symptoms which are met with in cerebral hæmorrhage, embolism, and thrombosis, and the softening which follows these lesions.

In *cerebral hæmorrhage*, optic neuritis is extremely rare, there being very few undoubted cases on record. So rare is it that Gowers suggests that when met with it may be due to some co-existing lesion, rather than to the extravasation of blood. The suggestion is certainly justified, for other possible causes of optic neuritis are often present in cases of cerebral hæmorrhage. Some years ago Dr. Bristowe reported* a case occurring in this hospital, and it is one of the very few I know of in which no other explanation for the optic neuritis seemed feasible. The hæmorrhage occurred in the posterior part of the optic thalamus, and very careful post-mortem examination by Dr. Sharkey revealed no other cause for the optic neuritis which was undoubtedly present. In many of the reported cases of optic neuritis due to cerebral apoplexy, it is more than likely that the hæmorrhage has occurred either in or surrounding a soft, rapidly-growing cerebral tumour — glioma — which is known to be very prone to hæmorrhage. Such tumours are extremely vascular, and the hæmorrhage so masks the appearances of a new growth,

* "Oph. Soc. Trans.," vol. vi., 1886.

that unless very careful examination is made, the case is regarded as one of uncomplicated cerebral hæmorrhage. In view of these facts cases of apparent cerebral hæmorrhage accompanied by optic neuritis should be regarded with suspicion, as it is very probable that other lesions are present.

Retinal hæmorrhages are met with in a considerable proportion of cases of cerebral hæmorrhage. As might be expected, the condition of the small vessels or of their walls, which leads to extravasation in the brain, also leads to extravasation in the retina. Cerebral apoplexy, as you probably know, occurs most frequently in the subjects of granular kidney. These patients are not unlikely to have hæmorrhage from their retinal vessels, quite apart from the special form of retinitis associated with chronic renal disease.

The changes in the vessels which are the most prolific form of brain hæmorrhage are miliary aneurysms. These are frequently met with in the brain, but are uncommon in the retinal arteries; the reason for this difference is, however, not very obvious. A subject of retinal hæmorrhage, especially if senile and with degenerate arteries, is often subsequently attacked by cerebral hæmorrhage: hence retinal hæmorrhages, without obvious local cause, may be regarded as indicating the probability, certainly the possibility, of future cerebral apoplexy. Of course it does not necessarily follow that a patient having retinal hæmorrhage will die of cerebral hæmorrhage, but the connection has been so frequently observed, that retinal hæmorrhages, without some local disease to explain their occurrence, should always cause a suspicion that the cerebral vessels may subsequently give way, and death may ensue from the resultant extravasation in the brain.

In *cerebral embolism* and *thrombosis*, ophthalmoscopic changes are not very frequent. In cases of embolism of the cerebral arteries there may be a coincidence of embolism in the retinal arteries, either in the trunk of the arteria centralis retinæ or in some of its branches. In such a case, the ophthalmoscopic appearances leading to a diagnosis of retinal embolism would be valuable evidence that the cerebral lesion was also embolic. Among the drawings I pass round there is one of the fundus oculi of a man who attended at the hospital. There is, as you will see, plugging of one of the main branches of the central artery of the retina, as well as of one small branch. This patient came under observation on account of his

ocular symptoms; he had some months later symptoms pointing to localized vascular change in the brain, which was most probably embolic. It may have been hæmorrhage from a small aneurysm, but in view of the fact that he had embolism of his retinal arteries, it is probable that he also had embolism of some of the cerebral vessels.

In the softening of the brain consecutive to embolism, optic neuritis has been noted, and the cases have been recorded; but here, again, arises the question whether such may not have been cerebral new growths, around which softening and hæmorrhage had occurred. In some instances, however, the emboli have come from recent valvular disease of the heart, and under these circumstances it would seem reasonable to assume that these emboli, being perhaps septic, set up inflammatory changes at the point at which they became arrested, and that an extension of this inflammation may give rise to optic neuritis.

Thrombosis occurring in the cerebral vessels may be accompanied by thrombosis in the retinal vessels.

Although ophthalmoscopic changes in these cases of embolism and thrombosis are not very frequent nor characteristic (and therefore ophthalmoscopic examination may not be of much help in the diagnosis of the cerebral lesion), instances occasionally happen in which the ophthalmoscope proves of some service indirectly. Such are cases in which there are cerebral symptoms pointing to either localized or extensive disease of the small vessels of the brain, and in which examination of the eyes reveals evidence, which may be wanting from other sources, of former syphilis. In cases of softening from syphilitic disease in the cerebral vessels, old syphilitic changes in the eyes are not uncommon. A case which I saw here a few days since may, I think, be mentioned in this connection. The patient, a man about 50 years of age, was sent to the hospital by his medical attendant on account of attacks of giddiness and temporary failure of vision, unaccompanied by any other cerebral symptoms, and it was thought that examination of the eyes might throw some light on the case. No evidence of recent disease could be detected on ophthalmoscopic examination, nor was there reason to suppose that the attacks were ocular in origin. We did, however, find changes which were almost certainly due to former syphilis, and this seemed to suggest that the symptoms complained of might be due to syphilitic disease

of the vessels of the brain; at least, it furnished a definite indication for treatment.

We now have to discuss (1) symptoms due to interference with the muscles of the eyes—oculomotor symptoms—occurring in hæmorrhage or softening from thrombosis or embolism in the brain (and, as I mentioned in last lecture, sometimes caused by small new growths), and (2) disturbances of vision resulting from lesions of this kind—hæmorrhage, softening, etc.—in the centres of vision or in some part of the visual path between such centres and the optic discs. These may be much better localizing symptoms than those revealed by the ophthalmoscope, such as optic neuritis, retinal hæmorrhages, or disease of choroid.

In dealing with this part of our subject, I think it will be simpler if we take up the ocular symptoms in order, and try to satisfy ourselves as to the position of the brain lesions producing them, rather than to consider the diseases of the brain individually, and study the symptoms met with in connection with each. I will first point out that such local disease of the brain may produce ocular symptoms which are spoken of as either (*a*) direct symptoms, or (*b*) indirect or distant. There is often considerable difficulty in distinguishing between these two groups. By a direct symptom is meant one which depends upon destruction, or disturbance of function, of the part of the brain in which the lesion is situated—that is to say, it is a symptom due to the direct effect of the lesion upon the brain substance where it is placed. An indirect, or distant symptom (the term "distant" is preferable), is one which is dependent upon pressure by the lesion, or its interference with the circulation in neighbouring parts of the brain, not of the part in which the lesion is actually situated.

It is an interesting and curious fact that lesions in all parts of the brain may sometimes fail to give rise to ocular symptoms, although other cases will be met with in which lesions apparently similar in position and other respects will produce definite localizing ocular symptoms.

Let us first take the oculo-motor symptoms. One of the most striking of these is what is known as conjugate lateral deviation of the eyes, which, as you probably know, is deviation of both eyes in the same direction, either to the left or to the right. There may not be actual deviation, or deviation may be slight, but there may be loss of the power of rotating the eyes in a given direction, so as to

look either to the right or to the left. This symptom is met with in cases of cerebral hæmorrhage, or embolism, and may be due, first of all, to a lesion in the cortex, although we do not know exactly in what part of the cortex the centre controlling the conjugate lateral movement of the eyes is situated. It is met with in lesions in the internal capsule, and in such cases there is nearly always, in addition, hemiplegia, and not infrequently rotation of the head in the same direction as the deviation of the eyes; the hemiplegia will then be on the opposite side to that towards which the eyes are turned (right hemiplegia, eyes deviated to the left, lesion in left side of brain); as is commonly stated, the patient is unable to look to the paralyzed side. Some of the French writers put it, "The eyes look towards the cerebral lesion." In cases where this conjugate deviation is due to cortical lesion, it is usually very temporary, and this is thought to indicate that lateral movements by the muscles of both eyes are represented in both hemispheres, but chiefly in the hemisphere of the opposite side. When the lesion occurs, the hemisphere which is unaffected subsequently takes on the control of this action. This same conjugate deviation is sometimes met with in lesions in the pons varolii, involving the nucleus, which is common to the third and sixth nerves, distinct from the nuclei of origin of those nerves, and which is generally supposed to be situated in the olivary body. In cases where the symptom is due to lesion in the pons, the ocular symptoms differ somewhat from those caused by a lesion in the internal capsule, the difference being that in the former the eyes are turned towards the paralyzed side in destructive lesions, and away from the convulsed side in irritating lesions. As Gowers has pointed out, symptoms in lesions of the pons vary according to the involvement or not of the sixth nucleus. If the lesion is above the sixth nucleus in the superior olivary body, the eyes cannot be rotated towards the side of the lesion, but the optic axes remain parallel. If the nucleus of the sixth is involved, there is complete loss of power in the corresponding external rectus (with deviation of the eye inwards), and there is also loss of the conjugate action of the internal rectus of the fellow eye with the paralyzed external rectus. The presence of conjugate lateral deviation may be of some assistance in diagnosis between lesions of the cortex, or internal capsule, and lesions in the pons, and it may also help us to

determine the side on which the lesion is situated in cases where other symptoms are not available, as, for instance, in cases of hemiplegia with complete unconsciousness.

Now, a few words about the ocular symptoms in so-called crossed hemiplegia, caused by a lesion situated in the crus cerebri. In these cases there is paralysis of the third nerve (usually, but not invariably, in all its branches) on the same side as the cerebral lesion, and hemiplegia on the opposite side. If the lesion extends beyond the crus, there may be present, in addition to the hemiplegia, hemianæsthesia, and paralysis of the facial and hypoglossal nerves. The usual combination is hemiplegia on one side, and paralysis of the third nerve on the opposite side, paralysis of the third nerve being on the same side as the lesion in the brain. This combination is of localizing value only if the hemiplegia and paralysis of the opposite third nerve occur simultaneously. If they come on separately they may be due to two separate lesions.

Complete paralysis of the third nerve is almost always due to lesions at the base of the brain; and the same is true of the fourth and sixth. The sixth is more commonly paralyzed than any other cranial nerve; and this peculiarity is explained by most writers by the fact that it has a much longer course to run, and therefore is more liable to be pressed upon by basilar lesions of whatever kind.

Paralysis of the sixth nerve, simultaneously with hemiplegia of the opposite side, indicates a lesion of the pons on the side of the paralyzed sixth. Paralysis of the sixth or other oculo-motor nerve may mean very localized cerebral disease, such as hæmorrhage or embolism, if there are no concomitant symptoms pointing to an extensive lesion. The following may be such a case:—A lady, 70 years of age, came to me in August with diplopia. She had incomplete paralysis of the right third nerve, but showed no other symptoms at all. The diplopia, which was her only cause of complaint, came on rather suddenly about three weeks previously, after a long walk, which unduly tired her. The opinion arrived at by myself and her regular medical attendant was that she had probably a small hæmorrhage (possibly embolism), affecting the nucleus of the third right nerve. A few days later she was attacked by numbness and weakness of the right hand; three months after that she died of cerebral hæmorrhage. (No *post-mortem* examination.)

Next, we come to symptoms produced, not by

interference with the movements of the eyeballs, but by lesions in the centres of vision, or in some portion of the visual tract between the centres and the eyeball. The most important condition is hemianopia, which means loss of the corresponding halves (or of corresponding portions not necessarily exact halves) of the two fields of vision. This may be due to hæmorrhage or to thrombosis, or to embolism, or to the subsequent softening which follows these conditions, or, as already mentioned, to tumours. The only variety of hemianopia with which we need at present concern ourselves is that which is known as homonymous lateral hemianopia, in which the right or left half of each field is lost. The presence of this symptom must of course be established by examination, and such examination is useless if the patient is unconscious, or if his mental condition is such that his replies are unreliable. Hemianopia may be caused by a lesion in the cerebral cortex; and, if so, the lesion is probably situated in the occipital lobe. Clinical and pathological evidence, which is now extensive, points to the centre of vision being situated in the occipital lobe, chiefly on its medial surface. It is now generally held that the actual centre of vision is situated in the cuneus, somewhere near its apex. Ferrier's experimental evidence that the centre is in the angular gyrus has not been borne out by clinical observation. Hemianopia may also be caused by a lesion anywhere in the course of the fibres between the occipital convolutions and the decussation of the optic nerves, at the chiasma.

If the lesion is situated in the occipital lobe, hemianopia exists, and is unaccompanied by motor paralysis. The loss of a portion of the field of vision does not give rise to what is known as a positive scotoma. On perimetric examination, half the field is found wanting; but the patient has no sensation of darkness in that part, just as healthy people are not conscious of the blind spot in their field of vision. The gap in the field in hemianopia is therefore known as a negative scotoma.

Proceeding down from the occipital lobe, we come to the optic radiations of Gratiolet, fibres which pass forwards to the optic thalamus. You will find it commonly stated that a lesion in the optic radiations may give rise to hemianopia, although some doubt exists, especially from recent investigations, whether these fibres are wholly visual fibres.

It is said that hemianopia is caused by lesion in the posterior part of the posterior limb of the internal capsule, and hemianæsthesia of the opposite side usually accompanies it. If the lesion extends rather far forward in the internal capsule, motor paralysis (hemiplegia) also will be present.* Lesions in the anterior corpora quadrigemina or the corpora geniculata externa do not give rise to ocular symptoms by which we can diagnose the site of the lesion. Cases have been recorded in which hemianopia was due to lesion in the pulvinar.

Next we come to the optic tract. Lesion of the optic tract usually produces complete hemianopia. As you know, the fibres from the right optic tract supply the right half of each retina, and, consequently, subserve the left half of each field of vision. Therefore, destruction of the right optic tract must give rise to loss of vision in the left half of each field.

A question which at once arises is this: Can we distinguish between hemianopia due to a lesion of the optic tract and that caused by disease higher up in the visual path? There is one symptom which, if present, tells us positively that the lesion is in the optic tract, and that is the symptom known as the hemiopic pupillary reaction described by Wernicke. By this is meant loss of, or marked defect in, the contraction of the pupil when light is thrown upon the blind half of the retina. In a case of lesion of the right tract, the right half of each retina is insensitive to light, and if light is thrown on this half of the retina, either no reaction of the pupil is obtained, or else the reaction is very defective. If the other half of the retina is illuminated, the impulse passes by way of the left optic tract, which is intact, to the corpora quadrigemina, thence to the third nerve, and gives rise to contraction of the pupils. Consequently, if Wernicke's pupil-symptom is demonstrable, we are able to say definitely that a lesion must be present in the optic tract, or, at least, at some point in this visual path lower than the corpora quadrigemina. It is not always easy to obtain this reaction because of the difficulty of getting a sufficiently small pencil of light thrown upon the retina. We may not have to rely on this symptom, however, for in many cases of optic tract lesion there are concomitant symptoms pointing to disease at the base of the brain, such as paralysis of cranial nerves, especially those supplying the

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* There is very little post-mortem evidence as to visual defects due to lesions in the internal capsule.

ocular muscles. A lesion of the optic tract may be so placed as to involve the crus cerebri, and in such a case hemiplegia of the opposite side would be present. Psychical symptoms, *e.g.*, word blindness, which may accompany cortical lesions, are absent in cases of disease involving the optic tract. I have recently had a patient under care at this hospital in whom there was left homonymous hemianopia diagnosed as due to a lesion of the right optic tract; the hemiopic pupillary reaction was present, and there was also complete paralysis of the right third nerve. This latter symptom, although helping towards localization of the lesion, proved an interesting complication, because the right pupil being paralyzed, the hemiopic pupillary test could not be tried on that side. But the left third nerve was intact, and in this eye the hemiopic pupillary reaction was shown extremely well.

We now come to a group of diseases of the brain and spinal cord, in which the changes are chiefly or wholly those of sclerosis. The diseases I propose to speak about under this heading are :—*locomotor ataxy (tabes dorsalis), disseminated sclerosis (insular sclerosis), general paralysis of the insane*, and *lateral sclerosis*, or what is generally termed *primary spastic paraplegia.*

We can dismiss the last mentioned in a few words. Primary spastic paraplegia is, in itself, a rare disease, and ocular symptoms in connection with it are extremely uncommon. Optic atrophy has been noticed, but so rarely that it cannot be looked upon as having any regular connection with this disease: the pupillary reactions are, as a rule, normal. In general paralysis of the insane, the ocular symptoms are probably always determined by the particular area of spinal cord or brain which is the seat of morbid change. As you are aware, some cases are marked by very decided mental symptoms, with little or no evidence of disease of the motor or sensory tracts, whereas others have but few mental symptoms, but have very marked evidence of sclerotic changes in the motor or sensory tracts of the spinal cord or brain, or both. In these three diseases—tabes dorsalis, insular sclerosis, and general paralysis of the insane, optic nerve atrophy is met with, and it is usually considered to be a primary atrophy, that is to say, atrophy not preceded by optic neuritis. In tabes dorsalis, this symptom is very common, although the proportion of cases in which it occurs has been stated by recent writers to be smaller than has been

generally given in text-books. Leber gives 26 per cent., and Michel, another collector of statistics, only between 12 and 13 per cent.; Gowers says not more than 10 per cent.; Buzzard 15.3 per cent. Probably we should not be far out if we took the average of these percentages. Optic atrophy in tabes generally occurs early in the disease, before there is much loss of co-ordination, and it is a curious fact, which has been repeatedly pointed out, that cases in which optic nerve atrophy occurs, are not severe cases in other respects. The spinal cord symptoms often develop extremely slowly; sometimes they appear to become quite stationary, although the optic nerve disease progresses to complete blindness.

In insular sclerosis, optic atrophy is by no means uncommon, but it differs in several respects from that met with in locomotor ataxy. It is less regular and progressive, is often unilateral (at all events for a very long time), and if bilateral is marked by decided want of symmetry. The ophthalmoscopic appearances are often suggestive of a hyperæmic condition of the disc and nerve, and, according to Uhthoff, a very good authority, inflammation of the nerve occurs in not a few cases—a low form of neuritis, not one that would be accompanied by swelling of the papillæ. In 100 cases of insular sclerosis, Uhthoff found 48 in which the fundus was apparently normal, leaving 52 in which there were ophthalmoscopic changes in the optic nerve. That is a very large proportion, larger than the highest rate reported in tabes. Buzzard, writing on this subject in 1893,[*] mentions that in his experience optic nerve atrophy is considerably more common in disseminated sclerosis than it is in tabes dorsalis (15.3 per cent. in tabes, about 40 per cent. in insular sclerosis). That is a complete reversal of the opinion which was widely held comparatively recently. Not many years ago, at a discussion on this subject at one of the London societies, it was stated by an exceedingly good authority, that the presence of optic nerve atrophy without obvious local cause almost certainly meant locomotor ataxy, although the spinal cord symptoms might be very slight, or even entirely absent.

Ophthalmoscopic examination in these cases, even if optic nerve atrophy be absent, will sometimes reveal changes which may be of value, namely, evidences of syphilitic disease. Suppose a case of optic nerve atrophy with very dubious

* "Brit. Med. Journ." October 27th, 1893.

evidences of spinal cord disease. Undoubted syphilitic changes, even of very old standing, observed in the eyes, would be valuable evidence as to the probable nature of the disease of the nervous system. You are aware that neurologists incline to the belief " no syphilis, no ataxy," and I think they have almost come to the opinion that the same is true of general paralysis of the insane.

Then with regard to oculo-motor paralyses, paralysis of the external muscles of the eye. These are common in *tabes dorsalis*, occurring as transient paralyses lasting for a few days, or weeks, or as a permanent defect. It is usually paralysis of one nerve, or possibly of one or two branches of a nerve. Transient ocular paralysis is said to be more common in the early stage of locomotor ataxy, and the permanent forms in the later period.

I have seen paralysis of the sixth nerve in tabes more frequently than paralysis of the third or fourth, but, according to records published some years ago, the third nerve is that most frequently affected, the actual figures being :—Third nerve 26 per cent., sixth nerve 12 per cent., fourth nerve 3 per cent.

In *disseminated sclerosis*, paralysis of ocular muscles is very uncommon, therefore the discovery of that condition may be of value in cases where there is some doubt between insular sclerosis, and sclerosis of the posterior columns only. Disturbance in the action of the ocular muscles, giving rise to nystagmus is however extremely common in this disease, and very rarely met with in locomotor ataxy, or general paralysis of the insane. The jerking movement of the eyes is usually from side to side, and is most evident when the patient tries to rotate the eyes to one or other side.

I will devote the few remaining minutes to the pupillary symptoms in these diseases. You are probably aware that in *tabes dorsalis* it is very common to have the condition known as the Argyll-Robertson pupil, that is a condition characterised by loss of the pupillary light reflex (contraction on exposure to light), the associated contraction of the pupil being retained. Usually, the loss of reflex contraction to light, is combined with loss of reflex dilatation on shading the eye. In a fair number of cases, we find " spinal pupils," a condition in which the pupils are unduly small, and may or may not have lost their light reflex. The Argyll-Robertson pupil was thought by the discoverer to be due to a lesion high up in the

spinal cord, in what was called the cilio-spinal centre. Later evidence is against that view, and makes it much more probable that it is due to degenerative changes in Meynert's fibres, which run from the corpora quadrigemina to the third nerve nucleus, and which form part of the nervous arc concerned in this reflex act. On exposure of the eye to light, the impulse travels up the optic tract to the corpora quadrigemina, and thence to the third nerve nucleus, and passes down the third nerve to the iris. If there is an interruption at any point between the corpora quadrigemina and third nerve nucleus, the impulse cannot reach the nucleus, and hence no impulse is sent to the sphincter muscle of the iris. Inequality of the pupils is not unusual in tabes dorsalis in addition to the other pupillary symptoms I have mentioned.

In *disseminated sclerosis* pupillary reactions are seldom disturbed, although inequality of pupils is not uncommon.

In *general paralysis of the insane*, pupillary symptoms vary very much. If the case is one in which there is evidence of disease in the posterior columns of the cord, we should expect to find pupillary symptoms, similar to those met with in ordinary cases of locomotor ataxy. A varying degree of inequality of the pupils is common in general paralysis of the insane. In the early stages, the pupils are generally small, and in the later stages dilated; in a great many instances they become unequal, and maintain their inequality throughout the disease.

Our time is now exhausted, but before we disperse, I should like to add that our subject is too wide to have been fully dealt with in the time we have been able to give to it, and hence in these two lectures, much that I would like to have spoken about has been necessarily passed by unnoticed.

Acute Catarrh.—

R. Sulphanilic (paranalino-
   sulphonic) acid   ... ʒ2½
   Bicarbonate of soda ... ʒ2⅛
   Distilled water   ... fℨ6½

Give 1¼ to 2½ fl. oz. daily, if necessary. No untoward effects, even when used for weeks. In doses of ½ to 1 drachm of the acid, action manifested within two hours, persisting twenty-four to forty-eight hours, when dose should be renewed.

(*Nouveaux Remèdes.*)

CLINICAL NOTES ON CASES IN CHANDOS WARD, CHARING CROSS HOSPITAL, FEB. 7, 1895.

By AMAND ROUTH, M.D., M.R.O.P.,

Assistant Obstetric Physician to the Hospital.

(By kind permission of DR. WATT-BLACK.)

Case 1. *Metritis, Salpingitis, Perimetritis.*— This is a young woman who has been married twelve months, but has not conceived. She is distressed at this fact; but her most serious trouble is that since her marriage she has suffered from irritating leucorrhœa and an increasingly severe type of dysmenorrhœa, which begins at least seven days before her "period" arrives. Her menstrual loss is profuse, lasting seven days, and the dysmenorrhœa continues till about the fourth day, when the flow becomes copious. Occasionally she has a very severe loss on the fourth or fifth day, amounting almost to a "flooding."

As a result of these troubles she is emaciated, nervous, and anæmic. She states also that she cannot sit or stand without discomfort, and that married life is agony to her, and causes severe abdominal and loin pain next day. There is only doubtful history of any acute attack of pelvic pain.

On examination the uterus is found hard, tender, and semi-fixed, and the tubes are swollen and low and tender. She has, therefore, the physical signs of the result of an acute or sub-acute metritis, salpingitis, and slight consequent perimetritis.

These conditions, which may be due to gonorrhœa, explain all the symptoms, and also the position of the pain in the loins. Tubal pain is sometimes referred to the hip-joint, sometimes to the sacro-iliac synchondrosis, but more often to the iliac crest posteriorly or to the kidney region, extending sometimes as high as the shoulder-blade, simulating renal calculus or pyelitis, or even pleurodynia.

This case may require removal of the appendages, but as the endometrium is here also greatly inflamed a preliminary curetting may be tried after all acute symptoms have subsided, with subsequent free antiseptic drainage by iodoform intra-uterine tamponning. Small doses of mercury, arsenic, and cannabis indica will simultaneously be useful, with blisters over the painful nerve areas.

Cases 2 and 3. These are cases of *venous thrombosis*, but differ in many respects.

In *Case* 2 the woman was confined five months ago, and had some swelling and œdema of the legs during the latter months of pregnancy. This subsided during the lying-in fortnight (during which more blood was lost than is usual), and, after being about a few days, she noticed renewed swelling of the left leg, followed in a few days by similar swelling in the right leg. The swelling was mainly below the knees, was accompanied by no groin nor pelvic pain, nor was there any pyrexia; well-marked pitting was observed, and the femoral and popliteal veins were found tender, cord-like, and knobby. There was no albuminuria, and with rest in bed, iron and purgatives, she has become quite well, and she will shortly leave the hospital with elastic stockings.

Here the thrombosis was due to pre-existing varicose veins, and was excited by the normal altered state of the blood, which contains, relatively, more water, less albumen, and more fibrin, thus predisposing to coagulation, and also by the profuse lochial loss during the lying-in period. There was at no time any pelvic inflammation, nor were the lymphatics involved, as in the next case.

Case 3 was a case of true *phlegmasia dolens*, both the veins and lymphatics being involved in what was primarily a septic pelvic inflammation.

There is a history of an abortion, eight and a half years ago, with pelvic inflammation following, and recurring attacks since, and resulting sterility. Her last attack was six months ago, during menstruation, ushered in by rigors, high fever, vomiting and constipation, and iliac pain and swelling. She recovered partly from this, but a few weeks after she had a sudden sharp pain in the right groin, which rapidly swelled, and in a few days became tense, shiny, white, and tender. The swelling spread rapidly down the leg, and there was simultaneously increased pain and tenderness over the ovarian region. On examination it was found that the uterus was fixed and pushed to the left by a mass which was apparently behind the right broad ligament, and there was also some distinct involvement of the lower lying cellular tissue round the right side of the vagina.

Here the leg state was secondary to the blood state associated with recurring peri- and para-metritis, with probably a chronic salpingitis, as the central pivot upon which the recurrences depended. The iliac veins and Broad Ligament veins and

lymphatics were early involved, and the leg was secondarily implicated.

Rest, soothing applications, purgatives, quinine in first stage and iron more recently, have nearly restored her to health, though the leg still remains large and somewhat numb.

These cases show that every swelling of the legs after labour is not necessarily phlegmasia dolens, and that, on the other hand, it is possible to get a typical phlegmasia dolens apart from a puerperal state.

Probably all cases of phlegmasia dolens—certainly those I have seen, are septic in origin. The first patient I saw with this disease, apart from pregnancy, was a case of dysmenorrhoea, in which I dilated, leaving one of Meadow's glass stems in the uterus. She recovered, lost her dysmenorrhoea, and then neglected to syringe. The result was that some septic material collected around the base of the stem; three months later she got a little pain in the pelvis, the uterus became tender, and there was evidently a little septic metritis. The doctor removed the stem, and a day or two afterwards she had acute mischief round the left tube in the peritoneal cavity. The following day, while moving in bed, she had sudden acute pain, and a typical attack of phlegmasia dolens.

Case 4. Fibroid—Abortion—Subinvolution.— This patient is 42 years of age, has had eight children—the last two and a half years ago, and had an abortion fourteen months ago. There was nothing peculiar about her catamenia. She dates her trouble from a miscarriage she had in November, 1893. She got up on the fourteenth day, and since that time has been complaining of trouble—bearing-down pains, pains in the back, and excessive loss of blood during menstruation. There has been first a yellow discharge tinged with blood, and the discharge has been aggravated by exertion. The sound passes $3\frac{3}{4}$ inches, but the uterus is distinctly larger than this would imply, and larger than it ought to be in a case of pure subinvolution of the uterus. With the aid of the sound and as a result of bimanual examination, it is easy to make out that there is a fibroid of the uterus, in the fundus, bulging down backwards. The lining membrane was felt to be uneven, and was very vascular, bleeding freely. The history seems to be that since she had her last child, two and a half years ago, a fibroma has been forming with some chronic endometritis, and that this led to the

abortion in November, 1893, and subinvolution ensued, the presence of the fibroma preventing the proper involution of the uterus, the persistent hæmorrhage being caused by chronic and decidual *débris* and by the co-existing endometritis. The uterus was rapidly dilated and curetted a few days ago, and the hæmorrhage has already nearly ceased. Ergot has been continued, and if it were advisable to pass the sound, the uterus would be found to be reduced in size, though it will not of course involute normally, owing to the interstitial fibroid.

Case 5. Chronic Endometritis with Subinvolution, Septic Salpingitis, Perimetritis.—This woman, aged 42, has been married twenty-six years, and has had fifteen children, the last one having been born last July. The case somewhat resembles the last in there being evidence of subinvolution with endometritis after this last confinement. There was pelvic pain, backache, bearing down, and somewhat offensive and often slightly pinky leucorrhoea. She was therefore in just that state which is most liable to suddenly develop into an acute peritonitic inflammation on the smallest provocation, such as an over walk, a slight chill, the passage of a sound, or a menstrual period; or such a complication may occur without any such cause being discoverable.

Either by a septic infection or by a spreading of the inflammation from the uterus, the tubes become involved, and then the whole pelvis behind the broad ligaments may become hard and solid by exudation of lymph and matted bowel, the matting rising sometimes into the iliac and hypogastric regions. This has occurred here, the uterus being quite fixed, and its size and shape being for the time being entirely obscured. The temperature varies from 101° to 103°, but there are no night sweats, and it is now showing a downward curve, the pain is less, there is no point of softening, the matting of the bowel is less marked, and the exudations felt *per vaginam* are more nodular and harder, showing signs of becoming absorbed, so that we may expect her to get well without suppuration occurring.

Antiseptic douches, free bowel action, perchloride of iron as a blood tonic are the measures found useful, with fomentation to relieve pain, and blisters to check inflammatory extension and encourage absorption.

This is one of those cases where the passage of a sound might and often does cause a similar

inflammation. And it is far safer, when in doubt, to dilate and explore the uterus under anæsthesia with antiseptic precautions, than to casually explore a uterus in such a condition as this was before the acute inflammation occurred, by means of a uterine sound. An abrasion may be caused, and septic absorption and its results speedily follow.

Case 6. Albuminuria of Pregnancy (3½ *months*). *Induction of Abortion. Recovery*.—This patient, aged 26, has had four children, the last four years ago. She has had a trying time in the hospital in different ways. She came in last November, into the medical ward under Dr. Green, being then three or four months pregnant. She came in on account of swelling in the face and limbs, nausea, and morning headache. On examination, it was found that the urine was acid, sp. gr. 1023, and that it contained about one-third albumen. Dr. Green diagnosed acute nephritis of pregnancy. There was blood in the urine, and I believe there is a trace of it even now; there certainly was yesterday: and the urine, of course, contains a trace of albumen. Dr. Green believed there had been no chronic nephritis previous to this pregnancy, there were no granular casts, no evidence of very high arterial tension,—judging from the pulse, and from the eyes. She got worse and worse, had vomiting, giddiness, constant headaches, epigastric pain, everything tending to one of the conditions supervening upon that of albuminuria; I mean such conditions as loss of sight, deafness, facial or other forms of paralysis, such as hemiplegia, or paraplegia, melancholia, or, what was more likely, eclampsia. But I cannot find any notes as to how much of the albumen, which was precipitated by heat and nitric acid, was paraglobulin, and how much was serum. It depends very much on what proportions these occur in; as a rule the more paraglobulin the less serious is the prognosis. The relative amount of urea was much diminished, and this is one of the most important indications of further complication being impending. It was decided therefore, to bring on abortion, though this is not usually required between the third and fourth months. Dr. Black did this in the usual way, of course antiseptically, namely, by passing a sound into the uterus, without previous dilatation, separating the membranes from the uterine wall with the sound, trying not to rupture them. That was on January 16th. Antiseptic douches were continued, and pains came on in about 36 hours, and the following day the fœtus was born, but the placenta and membranes did not come away until 24 hours after that. No attempt was made to dilate the cervix and to remove the placenta, as there was no hæmorrhage, and everything was kept antiseptic. After the passage of the placenta, there was however a slight rise of temperature, and the uterine cavity was irrigated with a 1 in 3000 solution of corrosive sublimate, after which the temperature dropped immediately. The slight rise was undoubtedly sapræmic in nature.

One's great object in bringing on an abortion before the third month is to keep everything entire, so that it may come away *en masse*. It may be necessary to accelerate matters with sea tangles, dipping them into an alcoholic solution of corrosive sublimate, or if hæmorrhage be profuse or signs of sepsis occur it may be necessary to dilate up rapidly with bougies, get one's finger in, separate and remove everything. Here pregnancy was advanced nearly four months, and the ovum failed to come away *entire*.

It often happens with patients who have got over their main trouble, that the urine remains albuminous for a very long time, or a woman may have recurrences, and gradual development of chronic nephritis. Happily there is every reason to believe that this patient will get completely well, though the house-surgeon tells me there is still a little blood to be found in the urine occasionally.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Tuesday, March 19, 1895.

The Most Superficial Form of Rodent Cancer.

A woman, aged 60, sent by Dr. Stocker, of Forest Gate, showed an extremely superficial form of rodent cancer in a somewhat unusual position. She had a patch of scar as large as a watch face on the left side of her chin. It was of irregular shape, and had advanced almost to the red edge of the lip. With the slightest possible exception

this scar was perfectly sound, and at its edge there was nothing to be seen excepting a very narrow, sinuous roll of induration, scarcely thicker than a stout pin. The disease had been present for nearly ten years, and was slowly advancing. A sister of the patient was believed to have died of internal cancer.

Comments. This patient shows the most superficial form of rodent ulcer which I have ever seen, but the disease is quite characteristic. The sinuous rolled edge, although almost microscopic in the minuteness of its development, is unmistakable. The disease would appear to have travelled over the skin without causing ulceration, and leaving behind it a healthy scar. The patient tells us that at times, in connection with the weather, there has been a little transitory inflammation, but nothing of any moment. Itching has been the chief trouble. I have no doubt that the characters of the rodent form of epithelial cancer receive some modification in connection with the part of the face on which they originate. The disease is very rare below the level of the mouth, and in one or two other cases in which it occurred on the neck and upper part of the chest I have seen it attended with almost as little tendency to ulceration as in the present instance. The diagnosis in the present case might, however, easily be missed, for unless you look carefully you will see nothing but healthy scar. I will by no means assert that these unusually superficial forms of rodent are met with only on the lower part of the face, for several drawings in the museum show them on the temple and scalp. A lithograph given me by the late Mr. Cæsar Hawkins shows a large extent of thin scar on the temple of an old man, and with a very low rolled edge, conditions reminding us of what we have just seen in our patient.

Lupus of one Lower Extremity with Elephantoid Hypertrophy.

A very pale, delicate-looking girl, aged 15, was brought by Dr. Barnard, Mr. Hurry Fenwick's house-surgeon at the London Hospital. Her left lower extremity from the knee downwards was involved in ulceration and scar. The foot was much enlarged and clubbed, being in a condition of elephantoid hypertrophy, whilst most of the toes were shortened by interstitial absorption of

bone. The other leg was quite healthy. Above the knee there were several deep scars, which involved the cellular tissue and had resulted from subcutaneous abscesses. Similar scars, all of them deep, were present on both upper extremities. There was nothing in the patient's physiognomy or teeth in the least suggestive of hereditary syphilis, nor were there any facts in the family history that pointed in that direction. The patient's grandmother and two aunts had died of phthisis. The history of the disease in the girl was that it had begun at the age of 2 years by "lumps on the arms," and that subsequently others had formed on other parts. It appeared that she had been the subject of strumous abscesses from the age of 2 to 10, and there was a history of a severe attack of ophthalmia.

Comments. In illustration of this case, I have to show a photograph which exhibits the whole of one lower extremity affected by lupus, with elephantoid hypertrophy of the foot. Such cases are indeed not very uncommon. I show also another, illustrating a much rarer condition, in which the whole of the forearm and hand had been reduced by the ravages of lupus to a condition of scar, and all the digits shortened by absorption. Of this I have seen but two examples. In our patient's case, I do not for a moment suspect any taint of syphilis. The case is one of aggravated struma. You will note that the disease of the skin does not appear to have been exactly common lupus. The scars, of which the patient shows so many, none of them exhibit any lupus structure remaining, nor are they at all like the scars of lupus. The disease which has left them no doubt began in the subcutaneous cellular tissue, and not in the skin itself. On the leg, however, it affects the skin, and more closely approaches the ordinary conditions of lupus. But after all, lupus vulgaris and scrofula affecting the subcutaneous tissues differ rather in the location than in the nature of the morbid process. They are alike strumous and equally in connection with tuberculous antecedents. When, however, the disease has begun in one or the other form it usually keeps to it, and thus we usually see either multiple strumous abscesses or multiple lupus patches, and not the two together. This, no doubt, is explained by the law of contagion, to which we are so often obliged to have recourse, under which the products of inflammation affect chiefly tissues of the same nature as those in which they originated.

The following were some of the more interesting cases demonstrated on Tuesday, March 26th:—

Numbness and Pain in the Fingers in connection with a Palmar Ganglion.

In this case a woman, aged 35, who was brought by Dr. Tuchmann, had applied for advice on account of numbness of all the fingers supplied by the median nerve. The anæsthesia was not complete, but it appeared to be quite definite, and she easily recognized the difference between the two sides of the ring finger, the ulnar nerve territory not being affected. She complained, also, of very troublesome aching in the hand, and said that she was obliged to keep the fingers bent into the palm and to flex the wrist in order to relieve the pain. The cause of these symptoms appeared to be a ganglion in the front of the wrist. There was no very obvious swelling, the forearm being rather stout, but on careful examination there was not the slightest doubt that deep-seated fluctuation was present. It was also thought that by firm pressure some slight swelling could be produced in the palm of the hand. The patient gave no history of rheumatism nor of any special occupation which was likely to have caused the effusion.

Comments. I do not think that there can be any doubt that the pain and numbness in the fingers of which this patient complains are really due to the pressure of a tense ganglionic swelling upon the median nerve and other structures. Yet, as you have seen, the ganglion is by no means large, and it might easily have been overlooked. We are familiar with the ganglion in this position. It is often attended by distinct swelling, both in the palm and the front of the wrist, with communication under the annular ligament. It is often very much larger than in the present case, and may much disable the hand by making it feel weak and more or less painful in use. In my experience, however, it is not usual for it to be attended either by numbness or persisting pain. These peculiarities in the present instance (and they are the symptoms which brought the patient under care) are probably to be attributed to the fact that the parts have not yielded so much as is usual, and that the ganglion remains very tense. Although the patient gives no history of rheumatism, I should still suspect that the effusion is in association with gouty tendencies. Treatment should be by attention to diet, etc., in the anti-arthritic direction, and by the local use of blisters.

Very Large Patches of Lupus Vulgaris on the Buttocks and on One Leg.

This patient was a lad of 16 who was brought by Mr. Bland Sutton. The question of diagnosis was as to whether it should be regarded as lupus vulgaris or as a syphilitic form. A patch of enormous size occupied almost the whole of the left buttock, and was still spreading irregularly in various directions. Near to its borders were many satellite patches. Another very large and similar patch covered the front of the right knee. In both, scar tissue was forming in various places. The lad had opacities on both corneæ, and a swelling of some size freely fluctuating on his forehead. As to the duration of his lupus patches he could give no information. They had been there as long as he could remember. Neither in teeth or physiognomy were there any indications of inherited taint.

Comments. I feel no hesitation in, regarding the case as one of lupus vulgaris, and as having no association with inherited syphilis. The lad presents not the slightest indication of the latter. The opacities on his corneæ are like those left by strumous ulcers, and not those of interstitial keratitis, whilst the swelling on his forehead is, I think, a cold abscess, and not a node. As regards diagnosis by the appearance of the lupus patches themselves, I do not believe that it is possible, for in many cases of the lupus of syphilitic patients, it very exactly resembles lupus vulgaris. I cannot however recognize any form of chronic and persisting lupoid inflammation as occurring in the subjects of *inherited* syphilis. It is common enough after the acquired disease, but nothing is less frequent in association with the inherited. There is nothing in the appearance of the patches in this case, which in any way differentiates them from lupus vulgaris, and their chief peculiarity consists in their enormous size. This is due to the fact that they began in very early life, and have received little or no treatment. I will ask you to observe again, as we have so often remarked before, that the two patches appear to have begun simultaneously, and that they have not produced any others, excepting of course the satellites.

Warts as a Sequel of Syphilis.

A patient was brought by Mr. Hitchins, in whom, after both a chancre and gonorrhœa, there

were a crop of small warts in the sulcus of the prepuce.

Comments. I am glad to have the opportunity which this case affords for saying that I am a firm believer in the doctrine that syphilis causes papillary vegetations. We must admit, at the same time, that the irritation of gonorrhœal discharges, acting as other sources of irritation, may also induce them. My experience in practice is, however, that a majority of the patients who show a crop of warts in the sulcus of the prepuce, have previously suffered from syphilis. I believe further that in a great many cases these growths may be influenced by mercurial treatment. It is a very interesting fact, that a blood-poisoning such as that of syphilis may be the means of causing, not so much inflammation, as true overgrowth of normal structures, and it is quite possible that what we see proven in the case of the papillæ, may be true of other structural elements of the body. What then is the proof that syphilis can cause overgrowth of the papillæ? The best instance that I know, is the growth of a patch of warts in the middle of the dorsum of the tongue. This, I admit, is not of very common occurrence, but it is a very definite one. I have several drawings which represent such growths. They occur just in that part of the tongue which, when the organ is at rest in the closed mouth, does not touch the palate. They are not condylomata, but branching warts. I cannot conceive that they are due to any other influence than the syphilitic poison present in the blood, and so far as I have observed, they always occur coincidently with the syphilitic eruption. It is, of course, very common to see warts under the prepuce under similar circumstances, but when they occur in this position they cannot be claimed as proving the influence of syphilis, since they may be due merely to local irritation. This suggestion, however, will not apply to the case of the tongue. On the latter position I have seen them wither away under the influence of a mercurial course, and independently of any local treatment. Although I assert that these syphilitic warts are different from condylomata, yet we must remember that the condyloma is, after all, only a modification of a wart, and as much proves the tendency of syphilis to cause overgrowth as does the wart itself. The condylomata are papillary growths assuming peculiar characters in connection with the part affected and the patient's idiosyncrasy. They are seen for the most part only on the female

genitals, at the anal orifice, and in the mouth. Thus it seems evident that the moist secretions of the part have much to do in favouring their growth. It may be mentioned as a curious fact that they are seldom or never seen under the prepuce.

Syphilis in a Boy with large Condylomata around the Anus.

A boy, aged 11, sent by Mr. James Startin, was covered with the stains of a receding syphilitic eruption, but the chief interest in his case was that he had enormous masses of condylomata at his anus.

Comments. I will first ask attention to the peculiar localization of the syphilitic eruption in this case. It is very abundant and very conspicuous, but it is almost wholly confined to the limbs. In the expression, "the limbs," I include the haunches and the shoulders. The boy's buttocks and scapulæ, as well as the limbs, are covered with the stains, but the chest, abdomen, sides and middle of the back, are quite free. This arrangement is very peculiar, for, as a rule, syphilitic eruptions show themselves first on the trunk. I have, however, seen many other examples like the present one, and restriction to the limbs is a conspicuous feature in not a few cases of chronic non-syphilitic eruptions, which usually affect the trunk also. What the law is which effects this curious localization I do not know.

Next, as to the condylomata. We could not possibly have a better demonstration of what a condyloma is. All round the anus and adjacent parts there are smooth-topped, abruptly-margined, elevated patches. On looking carefully at these we see a number of red points, the tops of overgrown papillæ, but these are all welded together by a semi-transparent substance like bad size. We can separate the condylomata into several quite distinct masses, although they touch each other, and they are outgrowths on the skin, and not infiltrations of it, for the skin upon which they stand shows no evidences of inflammation.

We must now discuss the question as to how this boy has got his syphilis. No chancre has been discovered anywhere, and you see that his penis is small and quite free from irritation. We must not jump to the conclusion that because the condylomata are at the anus they indicate the site of the primary sore. They are constitutional symptoms and occur without any relation to the position

of the chancre. I fear, however, that it is most probable that this was the site of the chancre, although there is no proof of it. There are enlarged glands in both groins, and when these are found it becomes almost certain that the infecting sore was either at the anus or on the genitals. A case, which I saw a few days ago, afforded important evidence on this point. A young man, who was covered with a syphilitic eruption, made no difficulty at all as to admitting the nature of his disease, "but," he added with some surprise, "I have not had any chancre whatever." He had hard bullet glands in both groins, and, as I could find no trace of irritation on his penis, I could but suspect that he had a sore in the urethra, although there were no indications of such. Fortunately he gave me a hint by telling me that he had "a pile." On examining the so-called pile, I found a very well characterized and very hard chancre. It was limited to one side of the anal orifice, and projected from it to a height of at least half an inch. Its surface was perfectly clean. I am glad to say that I have seen exceedingly few cases of chancre at the anus; but it is just possible that a few of the cases, of which I have seen several, in which no chancre whatever could be discovered, were of this nature. In the case before us an additional fact has to be mentioned, that it is known that the boy's elder brother, who has been sleeping with him, has suffered from syphilis.

Patch of Lupus on the Back of the Hand of a Girl.

The subject of this case was a girl who was brought for demonstration by Dr. Blake. The patch was on the back of one hand, and about the size of a shilling, and presented the warty edges and chronic ulceration which usually characterize lupus necrogenicus. It had been present for a long time, but there was no history of any injury.

Comments. The case is of interest chiefly because Dr. Blake has already cut sections and made a most careful examination in the search for the tubercle-bacillus. The results have been negative. I must contend, however, that this makes no difference in our diagnosis. We cannot wait for the demonstration of the bacillus in cases of lupus, for experience has shown that it is only to be found in a minority of cases. Yet we do not on this account abandon our belief that the bacillus plays an important part in the develop-

ment of all these chronic scrofulous inflammations of the skin. The more chronic the process, the less abundant, probably, is the parasite, and the more nearly does the process approach one of common inflammation.

THERAPEUTICAL NOTES AND FORMULÆ.

Blennorrhagic Urethritis.—Citric acid, $15\frac{1}{2}$ grains; water $3\frac{1}{4}$ fl. oz. Give in six injections daily. Recovery should follow on eighth day. Or, for lavage use, 2 drachms of citric acid to 1 quart of water once daily. Recovery occurs on fifth day at latest. Tried in fifteen cases without a failure and without orchitis appearing in any.

(*Bull. Gén. de Thérap.*)

Guaiacol.—Recommended in pultaceous angina, phlegmonous tonsillitis, etc., in which diphtheria does not play any *rôle*. Equal parts of glycerin and guaiacol for adults, two parts of glycerin and one of guaiacol for children. Paint affected parts four times in twenty-four hours, making the last application late at night and the first early in the morning. Fever rapidly diminishes, and food can be taken without pain.

(*Jour. de Méd. et de Chir. Prat.*)

Antiseptic Dentifrice.—

℞	Thymol	...	...	gr.ivss
	Ext. of cochlearia		...	f ʒj
	Comp. tinct. of melissa			f ʒj
	Tinct. of ratanhia		...	f ʒiiss
	Essence of caryophyllus			ℳxvss
	Essence of menthol		...	ℳivss

Use three drops in a quarter of a glass of water.

(*Med. Chir. Rundschau.*)

Icteric Pruritus.—

℞	Alcohol	...	...	f ʒ$1\frac{1}{4}$
	Sulphuric ether		...	f ʒ$1\frac{1}{4}$
	Ichthyol		...	ʒ$2\frac{1}{2}$

Rub thoroughly into the skin. Dissolves cholesterine crystals irritating the excretory canals of sudoriparous system, and prevents them from forming again by impregnating them with a resinous substance.

(*Jour. de Méd. et de Chir. Prat.*)

A CLINICAL LECTURE

ON THE

PATHOLOGY AND TREATMENT OF AN ORDINARY BILIOUS ATTACK.

By ROBERT SAUNDBY,

M.D. Edin., F.R.C.P. Lond.,

Professor of Medicine in Mason College; Physician to the General Hospital, etc., Birmingham.

GENTLEMEN,—In the course of practice you will see too many cases for which neither you nor anybody else will be able to do much; but, it is to be hoped, you will also meet with a fair proportion of curable diseases, in the treatment of which you will have a good opportunity of showing what skill you possess, and of earning the reputation upon which your future success will largely depend. A patient cured, *tuto cito et jucunde*, is an excellent walking advertisement of a perfectly legitimate kind.

Of all classes of curable diseases there is not one which affords more chance of securing a brilliantly successful result than a bilious attack, for in none are the symptoms, often severe though they be, so completely under the control of rational therapeutic measures, while a true conception of its pathology and a well-considered method of dealing with its symptoms are by no means universally diffused. Abernethy is traditionally reported to have said that he could only define a bilious attack as "that condition which was cured by blue pill;" and it is quite probable that he did say some such thing, for in his admirable essay "On the Constitutional Origin of Local Diseases," which every one of you should read, he describes with much detail and wealth of illustration a series of examples of bilious attacks, for which he prescribed, among other rational remedies, blue pill. But the physiology of digestion and the pathology of the stomach were, in Abernethy's time (his essay was published in 1804), but little understood, and what appeared to him merely a collection of symptoms depending upon deranged function is clearly to us the effect of catarrhal inflammation of the mucous lining of the stomach and duodenum.

In the speech of the people the word "bilious" is used very loosely; it signifies nausea, want of appetite, headache, or any other symptom which the particular individual speaking has learnt for any reason, rightly or wrongly, to attribute to a mysterious affection of his "liver," which he calls "biliousness." Often enough the symptoms have nothing to do with a proper bilious attack, but are of nervous origin. However, it is not my present purpose to enter into any discussion of the nervous relationships of gastric disorders, but I may mention migraine as an example which is constantly and most erroneously regarded as a "bilious" symptom. It is only natural that the public should make mistakes of this kind, but it is to be regretted that *the liver* should also still figure largely in the pathology of the family doctor. It is unfortunate, because it shows that he has not grasped the true nature of the mischief, and therefore is unable to see as clearly as he should do how to remedy it. "It is only your liver out of order, my dear fellow!" may be very reassuring, but it conveys an entirely false impression to the mind of the patient, and is presumably also the result of very inadequate pathological knowledge on the part of the doctor.

It is false, because it is not the liver but the stomach which is the primary seat of the disease, and it is inadequate because it entirely fails to represent the inflammatory process, too often the result of irritating ingesta, which is the pathological basis of the symptoms.

Any one who is told that he has an inflamed stomach will, if he has ordinary common sense, be careful what he puts into it; but the liver is a comparatively remote organ, and, as in popular pathology, its derangements are generally attributed to its own "sluggishness," analogy suggests the propriety of trying a little more of the same stimulus, which has, unhappily, been the cause of the whole mischief. Our organic sensations are, at least in this instance, very apt to be misleading. Witness the classical case of Alexis St. Martin, which I have quoted before; but it will bear repeating, as it illustrates more graphically than any

ordinary clinical description the facts I wish to impress upon you.

As is well known to most of you, St. Martin was a man with a large fistulous opening into his stomach, whom Dr. Beaumont retained in his service for the purpose of physiological observation. After drinking ardent spirits pretty freely for eight or nine days, he complained of an uneasy sensation with tenderness at the pit of the stomach, some vertigo with dimness and yellowness of vision on stooping and rising again. He had a thin yellowish-brown coating on his tongue, and his countenance was rather sallow, but he felt otherwise well; his appetite was good, and he slept as usual. This would be a mild bilious attack from the point of view of the patient. Now notice what was the condition of the stomach, as seen through the fistulous opening: "*The inner surface of the stomach showed extensive erythematous livid patches from the surface of which exuded small drops of grumous blood, large and numerous aphthous ulcers, the whole covered with thick mucus. The gastric fluids when extracted were mixed with a large proportion of thick ropy mucus and considerable muco-purulent matter, slightly tinged with blood, resembling the discharge from the bowels in some cases of chronic dysentery.*" What a terrible description of the state of a gentleman's stomach! Yet he was, apparently, not much out of sorts. We are told he had a good appetite; he felt well in himself, and would probably have made little or no modification of his ordinary habits in regard to food and drink. As far as words can help you to a vivid conception of the pathology of a bilious attack, you have that assistance in this description. Unfortunately, our patients do not afford us the same facilities for directly inspecting the state of their stomachs under these conditions, and that is my excuse for quoting and re-quoting this case. It teaches a lesson which we cannot learn at the bed-side or in the post-mortem room, although we often see in the last-named place the result of many such attacks in the familiar appearances of chronic gastritis.

Such an acute gastritis, varying only in degree, is set up, not only by the excessive use of ardent spirits, but by many other irritating ingesta, by catching cold, and by almost all infectious processes; and it is very important to remember that one attack predisposes to recurrence of the disease. People who have previously enjoyed the happiness of perfect digestion find, after going through some acute illness, such as typhoid fever, pneumonia, or influenza, that they have become liable to suffer from bilious attacks after slight errors of diet, from a heavy dinner, an unaccustomed amount of wine, a railway journey, or a common cold; and their welfare greatly depends upon their falling into the hands of a practitioner who knows what I have been telling you, and perhaps also who knows what I have yet to tell.

The treatment of a bilious attack can now be placed upon a rational basis, even though we have little to add to those details which Abernethy deduced empirically a hundred years ago. His method was excellent, and his success was great, but, partly because it was not based upon sound pathology, partly because it has been often applied, through faulty diagnosis, to unsuitable cases, it is half forgotten.

When we have to do with an inflamed stomach, the character of the diet, the ingesta, which, by their mechanical and chemical qualities, react upon the inflamed walls, will be admitted upon all sides to be of the very first importance. Common sense (or should we say ordinary surgical principles?) teaches us to keep those inflamed walls from contact with rough moving particles, to bathe them in bland fluids, and to establish a free drain, so as to prevent decomposing matters from remaining in their cavity. No doubt a diet of milk and lime water fulfils admirably the first and second of these indications, but it is not ordinarily necessary to restrict the patient to such a diet, which should only be ordered where vomiting or purging indicate an extreme degree of irritation. Like St. Martin, he often feels pretty well, and has a fair appetite, so we must not be unnecessarily rigorous, or we may forfeit our patient's confidence and drive him to a rival, who will "score off" our want of knowledge. We must stop all articles of diet which are not easily broken up by the teeth into a fine pulp. Here let me interject the remark that defective teeth are often the cause of bilious attacks. And we exclude all substances which contain irritating principles, such as pepper, and those which are prone to undergo decomposition in the stomach and form acrid products: for example, fats, starchy food, and sugars. As you may confidently expect a cure in three or four days, it is generally convenient to write out a diet for each patient. The effect of this is good, as it leaves no room for the insidious suggestions of

self-indulgence, which would easily drive a coach and four through your general directions. Therefore I recommend such a diet as the following :—

Breakfast : Cocoa, coffee, or China tea, with plenty of milk, but no sugar; a slice of broiled white fish; two small slices of dry toast; no butter.

Luncheon : Broiled white fish or lean mutton ; toast ; no vegetables; a baked custard pudding; no cheese. Drink claret well diluted with mineral water.

Dinner : No soup; white fish boiled or broiled without sauce; boiled or roast chicken, or mutton; toast ; no vegetables; jelly, custard, or isinglass blancmange. Claret and mineral water.

Cocoa is blander than coffee or tea, and China tea, on account of its much lower percentage of tannin, is to be recommended in preference to the teas of India and Ceylon. Fish should be broiled, not fried, as the outside of fried fish is tough, and charged with fat and fatty acids. Vegetables consist of starch, which is converted into sugar and readily undergoes fermentation in the stomach, and vegetable fibre which is insoluble by the digestive juices. Brown bread and porridge, which contain bran, must be forbidden, despite popular prejudice in favour of their "digestibility." Claret with mineral water, or any light wine similarly diluted, makes a harmless drink ; some patients like hot water with a slice of lemon in it; undiluted wines, and even diluted spirits, should always be forbidden although, again, in this last instance, we run counter to popular ideas, and, I regret to say, to very common medical practice. Bear in mind, as your patient advances towards convalescence, that any of the lighter meats, suitably cooked and carefully masticated, are very well tolerated by even an inflamed stomach, while starchy foods cause flatulence and other evidences of disorder.

We have, in these dietetic arrangements, sought to provide suitable nourishment without causing irritation ; we further desire to afford a drain to remove the products of digestion, so as to prevent their remaining unduly long in the stomach where they may undergo irritating decomposition. We can effect this by calling into play the normal mechanism by which the stomach, in health, empties itself at the right time, and this function is probably stimulated by the administration of blue pill better than by that of any other drug, though all aperients act more or less in the same way. The good effect of emptying the stomach down-

wards is too well known to need insisting upon, and usually it does all that is wanted, though there are cases in which emptying the stomach upwards by an emetic, followed by copious draughts of warm water, is a more speedy and more effectual remedy, though one not to everybody's taste.

I am in the habit of ordering two 5gr. blue pills, one to be taken on two successive nights, or omitting one night. In the morning, a breakfast-cupful of hot water in which a teaspoonful of Carlsbad salts have been dissolved, should be sipped slowly while dressing. Before each meal, the following powder should be taken suspended in a little milk :

R̶   Bismuthi carb. ...   ... gr.x
      Sodii bicarb.   ...   ... gr.x
      Pulv. rhei   ...   ... gr.ij
      Pulv. cinnamomi co. ... gr.v
Ft. pulvis.   Mitte xij

When the bowels are loose the Carlsbad salt may be omitted, and the hot water taken alone, and perhaps the rhubarb may be left out of the powder, but it should ordinarily be given, because, although patients dislike it, it is really useful, and taken as suggested in milk, it is scarcely nauseous.

I hope no one will complain if he finds this treatment at times unsuccessful in curing a supposed bilious attack, but in that case I would advise a reconsideration of the diagnosis. Is there not something more present? Above all, beware of the fallacious symptoms presented by gastric neuroses; and you will do well to look with suspicion on all supposed examples of recent gastritis, which present a clean tongue. In true bilious attacks, you will not find the principles and method I have laid down fail you in practice.

Chloral in Coryza.—Brodnax says that in coryza where the Schniederian membrane is very irritable, chloral gr.x (or drops); castor oil ℥ss used with a soft mop applied over the surface after being dried, acts to check the excretion of mucus, and lulls the irritation and the head pains.
(*Therap. Gas.*)

Laryngitis Stridulus.—Bromide of potassium in large doses, continued for several days. In a child of twenty-one months, the dose was gradually increased by the author until it reached 23¼ grains. Important to continue treatment for five or six days, to prevent return of the attacks.
(HUCHARD, *Jour. des Prat.*)

SOME CLINICAL OBSERVATIONS

ON THE

PRINCIPLES INVOLVED IN THE SURGERY OF FRACTURES.

Delivered at Guy's Hospital

By W. ARBUTHNOT LANE, M.S., F.R.C.S.,

Assistant-Surgeon to the Hospital.

WE will consider in order the various changes in the skeleton that may ensue in consequence of a fracture of one or more long bones; the unequal degree in which these changes take place in the upper and lower extremities; the factors determining their development; their clinical symptoms, the principles which should guide us in our treatment of fractures, so that we may avoid these changes altogether, or reduce them to minimum proportions, and the best manner in which these principles can be applied. I would remind you that the normal form of our bones and joints exists only as long as we lead a normal existence, combining various movements of activity with attitudes of rest. You are aware that if the growing subject is confined to attitudes of rest, avoiding movements of activity, deformities arise, which are due chiefly to changes in the joints and ends of the bones, as, for example, knock-knee; and again, if one movement, or sequence of movements of activity is assumed habitually, and other attitudes of activity with those of rest avoided, a deformity characteristic of the attitude of activity is developed, a deviation from the normal arising in consequence, being also due chiefly to changes in the articulations and in the ends of the bones.*

Obviously, then, our anatomy remains normal only as long as our mechanical relationship to our surroundings continues so, and any variation in the mechanical relationship is followed by definite physical changes in the bones and joints.

In other words, the problem of evolution is purely a mechanical one, and its proof is telescoped into the life-time of a labourer or into that of a child suffering from one of the resting deformities. These are the experiments that nature makes for us, and by their consideration we become able to understand larger problems.

. I think you will now agree with me that the form and structure of a joint remains normal only

* "A Clinical Lecture on Acquired Deformities," *Clinical Journal*, January 30, 1895.

as long as the mode of transmission of force through it remains normal; alter these lines of force transmission in any way, and changes in structure proportional in degree must arise.

Nature again makes experiments for us in which she alters, more or less completely, the direction and mode of transmission of force through one or more joints of a single limb. This comes about by the fracture of a long bone, whose fragments unite in such a manner, that the axis of the one corresponds no longer with that of the other, and as the angle formed by these axes with one another is often great, or if parallel, as they are frequently separated by a considerable interval, owing to overlapping of fragments, the mechanics of the joints formed by the ends of these bones, as of others influenced by the alteration in the lines of force, are changed in a definite and measurable degree from the normal.

Should we wish to study this subject further, it is obvious that the joints of the lower extremity offer us a much better field than those of the upper, for the reason that the weight of the body is habitually transmitted through the former, and in consequence the changes which develop in them must be very much exaggerated as compared with those which arise in the latter. Therefore we will proceed to examine the conditions of the joints of the lower extremity which result from fracture, and we will find that they may, for convenience sake, be divided into two distinct groups, though there is, of course, no sharp definition in the character of the changes. The very important factor which determines this variation in the results of pressure is the "age" of the individual, and its influence is one that must, as you will see, affect very materially the character of our treatment of fractures.

In the comparatively young adult considerable deformity, with the consequent alterations in the physiology or functions of the joints influenced, bring about changes which are, perhaps, best described by the term "adaptive." By this is meant necessary changes in the form, and, perhaps, in the planes of the articular surfaces without the articular cartilage being other than absolutely normal, together with a corresponding alteration in the limits of attachment of the ligaments and synovial membrane, both of which are normal in structure and function, while the secretion of the latter is not excessive in amount.

After a certain period of life, which varies

within wide limits with the habits, occupation, and surroundings of the individual, but which may be roughly averaged as about 40 years, this being, probably, much too high an estimate for labouring people, an altogether different series of changes takes place. These I have called "passive," for want of a better and more descriptive term. They consist in the removal of the articular cartilage at such places where the altered physiology of the joint causes the transmission of an amount of pressure which is abnormally great as compared to that which the same surfaces transmitted previous to the injury. After the cartilage is destroyed, the same factor, associated with un-accustomed friction, produces sclerosing, eburna-tion, and gradual removal of the opposing exposed surfaces of bone. The area and form of the articular surfaces are also modified by deposition of cartilage and bone on the margins, the synovial membrane becomes more bulky and develops abnormal fringes, and the synovial fluid is secreted in a quantity often largely in excess of what is normally sufficient. The ligaments may have their attachments displaced and their functions very materially altered, owing, to some extent, to changes in the axis around which rotation takes place. All these changes used to be regarded by the pathologist as indicative of the presence of a disease which was known by names such as rheu-matoid arthritis, osteo-arthritis, rheumatic gout, etc.; and he would be a daring student who, at an examination-table, would designate these changes as being other than due to this disease. Perhaps the best term to apply to them was one for which I am responsible,* namely, "mechanical or trau-matic arthritis," remembering, however, that these changes resulting from fracture form but a small group of that very comprehensive class.

As to the clinical manifestations of these condi-tions, the "adaptive" changes are generally un-associated with any pain or discomfort, except, perhaps, for a short time after the patient begins to get about, but the "passive" changes are on a very different footing. Their presence means that in their slighter degrees of development the patient experiences a feeling of insecurity in, or imperfect control over the movements of the part, and in their most marked forms a degree of pain that sooner or later absolutely incapacitates the sufferer from transmitting the weight of the body through the joints of the leg for any useful purpose.

* "Chronic Traumatic Arthritis," *Lancet*, January 30, 1892.

Nor is this the only trouble. Should the patient be affected with true rheumatoid arthritis, or with rheumatism, the chances are that the damaged joint or joints will be first attacked, or be more severely affected than others. Again, as all move-ments in the joints cause pain or discomfort, the patient uses the muscles of the limb in a very par-tial manner, retaining part or the whole of the limb more or less completely rigid. As a result of this, the foot, and later, the leg, become œde-matous, and organisms readily establish them-selves on such areas of the skin where the vitality of the tissues is most depreciated, and eczema, and later ulceration develop. As illustrations of the clinical conditions presented by the progressive development of "passive" changes, I will describe briefly two cases, not that they are remarkably severe, but because they represent very fairly the subsequent career of patients who sustain fracture of the long bones of the leg at or after middle life, the fragments not having united in a good posi-tion.

A. B., a powerful blacksmith, aged 40 years, was admitted into a general hospital in January, 1894, suffering from a fracture of the shaft of the femur about its centre. He was under the care of a very able and distinguished surgeon, who directed the leg to be put up in a plaster spica. He remained in the hospital nine weeks. I was asked to see him about the end of the year with a view to operative interference. The fractured leg was 2½ inches shorter than its fellow. On placing him on his back at rest the sound limb rotated outwards to its normal limit, while the inner margin of the foot of the damaged limb occupied a vertical plane. Owing to pain in the hip and knee joints, which was steadily increasing in severity, the man was unable to walk without a stick, and was, of course, incapable of following his employment. I did not consider that anything short of amputation would relieve him. To sug-gest amputation as the primary treatment for a simple fracture of the femur would appear ridi-culous, but the result of the usual surgical treat-ment of such a fracture may be so disastrous as to make the sufferer beg for it to be performed. Now, here was a patient placed under the most favour-able circumstances imaginable, yet what can be more deplorable than the result? and the same applies equally to the next case.

J. H., aged 49, a bricklayer's labourer, was ad-mitted into a general hospital in the early part of

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 9.
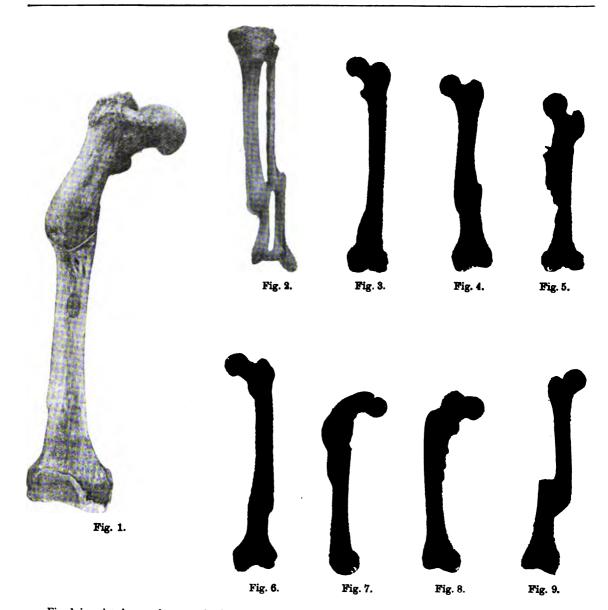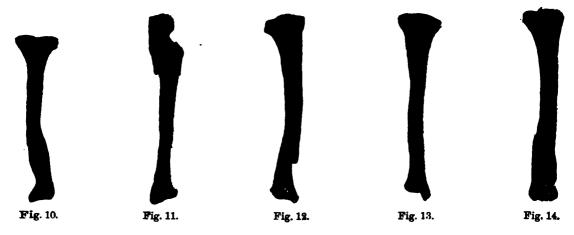
Fig. 1 is printed on a larger scale than the other illustrations in order to show in greater detail the several anatomical changes which take place in the joints because of the alteration from the normal manner in which pressure is transmitted through them in consequence of the all changed form of the bone, resulting from its fracture, and which are well marked in this particular specimen. Much of the deformity which exists has been produced by the leg and lower fragments having been rotated inward during the process of repair, to such an extent that the inner margin of the foot occupied a vertical plane, the rest being due to hæmorrhage into, and inflammation of the soft parts about the fracture, and the lower end of the upper fragment being of necessity forced outwards and forwards by the ascents of the lower fragment on its inner and posterior aspect. Observe the alteration in the form of the articular surface of the head pressure being transmitted through newly-formed bone outside and somewhat in front of the head ; also in that of the lower end of the bone where the disproportionate amount of weight sustained by the inner condyle has brought about extensive mechanical changes in it, the outer condyle being almost normal.

Fig. 2 represents a very common displacement in fracture of both the tibia and fibula. Observe that the lower fragments correspond to the vertically-placed foot, while the upper occupy the normal position of rest of the limb.

Figs. 3, 4, 5, 6, 7, 8 and 9, represent fractures of the femur in which deformity has resulted from the causes already described.

Fig. 10.   Fig. 11.   Fig. 12.   Fig. 13.   Fig. 14.

Figs. 10, 11, 12, 13 and 14 show similar conditions of the tibia and fibula. A careful examination of each specimen shows that this deformity, with the consequent physical depreciation could have been readily obviated at the time of the fracture by the simplest surgical procedures, it being hardly fair, perhaps, to signify the usual methods by such an attribute.

May, 1894, suffering from a Potts' fracture. A plaster case was applied, and he left the hospital on crutches ten days later. The case was removed in three weeks, and the patient hobbled about with the aid of a stick. He suffered so much pain in his foot that he sought admission into an infirmary, where he remained for several months. He recovered so far that he could get about slowly without a stick, though his foot caused him very great pain on movement. He had to give up his occupation of bricklayer's assistant as he could not climb a ladder, and as a general labourer he, with difficulty and in much physical distress, managed to earn a third of what he used to previous to the injury. The pain was situated chiefly in the subastragaloid joint. The seat of fracture is still evident as a depression, the displacement not having been reduced.

As an example of "adaptive" changes, I will merely relate one case, since it shows that such a considerable alteration in the mechanics of the joints resulting from the unusual deformity and shortening must have determined very marked deviations from the normal in their form and details. I was asked to see a lady of about 25 years of age, who some time before had sustained a fracture of the tibia and fibula below their centre, followed at once by great swelling, and who had been treated by back and side splints. Her medical attendant had the advantage of the assistance of a very able London surgeon in her treatment. I mention this not that I want to find fault with the treatment,

but on the contrary I wish to point out that by means of the usual methods it is obvious that nothing more could have been done under the circumstances. When I saw her the inner margin of the foot occupied the vertical plane, while its fellow included with it an angle of about 50°. The damaged limb was almost exactly two inches shorter than its fellow. She was a fine tall woman, and wished me to perform some operation by means of which her shortened leg could be lengthened.

Here, although there was this considerable shortening, and the lower fragments were displaced backwards and outwards behind the upper, she experienced no pain, discomfort, or insecurity in the use of the several joints. She wore a boot padded in its interior so as to compensate for much of the shortening, and this entailed a certain peculiarity of gait that caused her much mental distress.

Before passing on to the consideration of the mechanical principles that must guide us in our attempts to restore the fractured bone to its original form we must first investigate the obstacle which so often prevents this being effected.

We have, surrounding the long bones, a mass of muscular and tendinous structures, enclosed and bound down by more or less dense layers of fascia, which fit accurately over them in their conditions of contraction or relaxation. Around this there is a sheath of loose areolar tissue, which is covered in by the comparatively unyielding skin. If in the

dead body one of the long bones of the leg is broken through, and the fragments are displaced upon one another, it is possible, by traction and manipulation, to establish fairly accurate apposition. A very little experience of fractures in the living subject shows one that the conditions in it are altogether different, and a careful observer will notice that the difficulty presented seems to vary directly with the amount of hæmorrhage into and subsequent swelling of the soft parts around about the seat of fracture; also that, in consequence, if the fracture is situated in a part surrounded by an abundant quantity of soft parts, and the injury producing the fracture was so severe as to lacerate the tissues considerably and displace the fragments upon one another, very much more opposition is experienced to the approximation of the fragments than when the fracture occurs in a thin, feeble, old subject, with scanty, flaccid, soft parts, and in which the bone had yielded to very slight amount of force.

If you examine into the physical conditions, you see that the soft parts surrounding the long bone are ties in its length, and are practically incapable of extension. In a fracture the soft parts about the bone are lacerated so that the hæmorrhage is poured out freely into and beneath them, and this is encouraged by the overlapping of the fragments, which usually results from the force producing the fracture. Following rapidly on the hæmorrhage is a more or less active inflammatory process which involves the muscles, fasciæ, fat, and skin relaxed in the length of the limb by the overlapping of the fragments, which itself may even be increased by it to a considerable extent.

You can readily understand that such physical conditions as I have described render it practically impossible to place and retain the surfaces of a recent oblique fracture in apposition by traction, and that the restitution of the fragments into their normal relationship to one another is impracticable till the material which has shortened up the ties in the length of the fractured bone has been removed, and this cannot take place for many days.

The mechanical principles which must guide us in our treatment are, therefore, two, and here I may say that I am considering especially the peculiar circumstances presented by fractures of the lower extremity, though it is obvious that they apply with equal force to those of the upper limb also.

1. The axes of the fragments must be made to correspond as nearly as possible. This is effected by placing the patient in the supine posture at rest and by observing the foot of the sound limb, and measuring the angle included between its inner margin and a vertical plane, the degree of normal outward rotation of the thigh is readily determined.

By placing the foot of the damaged limb in exactly the same position the axes of the lower fragment or fragments will be made to correspond with that of the upper only when the opposition, produced by the shortening up of the ties in the length of the limb by blood and inflammatory material, has been eliminated by their absorption, sufficient traction being exerted in the length of the fragments in the meantime.

2. The second principle follows necessarily what I have already said, namely, that sufficient traction must be exerted upon the fragments in their length till absorption of the blood and inflammatory material has been effected to bring the broken surfaces into apposition. It is apparent that the amount of traction, together with the duration of the period during which it is exerted, must vary with the extent of hæmorrhage and subsequent inflammatory effusion, since in some cases while they are so small as to be practically absent it is possible, by exerting a moderate amount of traction and by manipulation, to reconstitute the bone in its normal shape, and to retain the broken surfaces in apposition by means of a plaster case, while in others the amount of hæmorrhage and inflammatory exudation may be so considerable, and their absorption so long delayed that union in an unsatisfactory position results. Though I had some idea that the influence exerted upon the ties of the long bones by hæmorrhage and inflammation was considerable, I never became alive to the immense resistance they offered till, in 1893, I cut down upon simple fractures of the tibia and fibula with a view of reconstituting these bones in their original form by uniting the fractured ends by steel screws.[*] In these cases I found that even after the skin and muscles were freely divided in order to expose the fracture, a very moderate amount of hæmorrhage and effusion was sufficient to resist what traction could be exerted upon the foot and lower fragments, so that it had to be supplemented by the use of elevators and lion forceps before it was possible to fix the broken surfaces accurately together with steel screws. It is of the truth of this

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[*] "A Method of Treating Simple Oblique Fractures of the Tibia and Fibula more Efficient than those in Common Use," *Trans. Clin. Soc.*, vol. xxvii.

that I have found most difficulty in convincing others; indeed it would seem that it is necessary for the surgeon to perform such an operation to realize this fact.

I do not think that I ever placed much reliance upon the considerable influence supposed to be exerted for some time upon portions of the long bones by muscles attached to them, and which is described diagrammatically and in detail in many of our surgical and anatomical works. Its influence in any particular case can be accurately gauged by administering an anæsthetic. Surgeons mistook this supposed factor in producing deformity, which in my opinion is practically non-existent, for the resistance offered by hæmorrhage into and inflammation of the soft parts about the fracture, and many lines of treatment and forms of apparatus were based on this false assumption. How this traction is to be exerted on the leg below the fracture must vary with its position. If the fracture is low down and near the ankle, and there is much swelling, it is obvious that but little force can be applied since the pressure that must be exerted locally by the extension apparatus upon inflamed and distended parts soon becomes unbearable. In the paper referred to I confined myself to the consideration of the treatment of fractures of the tibia and fibula, and I proved from a careful examination of a large number of cases in which such fractures had been sustained at or after middle life that, as regards the physical capacity of the labourer to perform his accustomed heavy work; or, in other words, his financial value as a machine, he was enormously depreciated under the methods of treatment at present adopted. This was naturally most conspicuous in such as were largely dependent on the perfect control of their lower extremities during the performance of their occupation, such as firemen, decorators, scaffolders, etc.

It would seem that, except in the case of fracture of the patella, this branch of surgery, namely, the treatment of fractures, has made no progress of any kind for a very long period of time; and even in fracture of the patella we find surgeons suggesting and making use of methods of a very imperfect character, rather than continuing the excellent and simple measures employed by Sir Joseph Lister. I said just now that the surgery of fractures has not progressed; I ought, instead, to have said that it has retrograded in a very marked manner, and this chiefly in consequence of the use of rigid cases which are applied

soon after the patient's admission. The best example of these is called Croft's splint, after the distinguished surgeon who invented it.

This method of treatment is both cheap and bad. It is cheap since it entails less expenditure on the hospitals, the patient being sent home at once or sooner than would have been the case if older modes of treatment were adopted; also the services of a mechanician are not required for the preparation of the splint.

It is bad since the presence of hæmorrhage and effusion in any quantity renders it impossible to replace the fragments at once in accurate apposition, and the fixation produced by the surrounding case ensures with more or less certainty, union of the fragments in an unsatisfactory position. In the less recent forms of surgery, when fractures of the tibia and fibula were treated by means of splints, there was more chance of there being less variation in the axes of the fragments since, in spite of the vertical footpiece the foot and lower fragments often rolled outwards, and as it was the rule to leave the seat of fracture uncovered, it was constantly under observation, and any marked deformity that became obvious on the subsidence of the hæmorrhage and effusion could be diminished by manipulation. Certainly, in general hospitals, the treatment of these cases rested to some extent with the surgeon, and was not left so much in the hands of his subordinates as it is at present,—as it is from necessity owing to the use of the Croft's splint. While I believe that a considerable number of fractures of the tibia and fibula occurring during or after middle life can be treated satisfactorily by extension, care being taken to ensure the symmetrical rotation of the fragments, I am even more convinced than I was a year ago that in oblique fractures of both bones especially when occurring somewhat low down, the wisest course to pursue is to cut down on the fractured ends, and by means of steel screws to retain the broken surfaces in accurate apposition, having first effected their approximation by traction on the foot and lower fragments, and by leverage exerted on the fragments by elevators, assisted with lion forceps or similar form of clamp.

In any case of fracture, in which the surgeon is doubtful whether he can bring and retain the fractured surfaces in accurate apposition, he should expose the fragments, and ensure a perfect result by the simplest of mechanical means. The greatest obstacle to firm union of the bones in this matter is extensive splintering of the ends, and

this sometimes renders the operation very difficult or even impossible.

In drilling the fragments the greatest care must be taken, and the surgeon, remembering that the barrel of the screw requires the construction of a hole of larger lumen than the part cut for the thread, should use a rimer for this purpose. If on driving home the screw, the resistance offered appears to be excessive, the screw should at once be withdrawn, and the hole enlarged.

The operation relieves the patient at once from the pain which results from the movement of the fragments on one another, and so removes any painful spasmodic muscular contraction, and it frees him from much of the tension and discomfort due to the presence of blood and inflammatory material in the soft parts.

How surgeons can remain satisfied with the results in these cases which statistics would seem to show they now obtain appears to me inexplicable, and I feel sure that they will soon realize the utter inefficacy of the modes of treatment still in use.

Passing on to the consideration of fractures of the femur occurring at the same period of life, namely, during and after middle life, I believe that the subsequent financial capacity of the individual is often depreciated to an extent not very far below, even if not in excess of, that which exists in fractures of the tibia and fibula, and this would appear to be due to the incomplete recognition of the two important principles which I have called attention to in this lecture, namely, the retention of the lower fragment in a degree of rotation, which is or which will subsequently become symmetrical with that of the upper, and the necessity of exercising traction till the ties in the length of the limb have reached their normal limit. Both these principles must be applied in the treatment, either alone is insufficient to obtain a perfect result. The physical conditions in the case of the femur lend themselves much more readily to treatment by mechanical apparatus alone than do those which exist in fractures of the tibia and fibula. Especially in fractures of the upper two-thirds of the shaft of the femur difficulties may not infrequently be experienced, which may seem to the surgeon to be insurmountable by these simpler means, and in such cases he may with advantage expose the ends of the fragments, and fix them accurately together by screws.

I have not investigated systematically the subsequent history of any long series of cases of fracture of the femur sustained by labourers during or after middle life, but I have examined very many who were depreciated mechanically to such an extent, that they could only get about with very great pain and difficulty, and were unable to follow their original active pursuits; and it is well to remember that once these conditions have resulted, it is practically impossible to remedy them by operative interference. Action must be taken before the fragments have united, or not at all. If operative interference is attempted after union has taken place in a bad position, it often involves such considerable risk as in many cases to render the operation hardly justifiable, particularly as it is impossible after the fragments have united to reconstitute the bone in its original form, since a considerable portion of its length comprising the overlapping ends must be cut away in order to permit of the sectional surfaces of the shaft being brought into apposition. This must be borne in mind by those surgeons who imagine that a mechanical failure having resulted, it is open to them at a later date to rectify the disastrous result by operative means. They may effect an improvement, but they cannot by any means, or by any operation restore the deformed bone to its perfect form, and the altering and painful joints to their original normal painless state.

Perhaps of all fractures of the lower extremity, none would seem to lend themselves more readily to operative interference than the condition which results from sudden and forcible excessive abduction of the foot, and which is usually described as Potts' fracture, since the broken bone is of small calibre, and is placed comparatively superficially. It is also dense in structure, and can therefore hold securely the wire or other material used to retain the fragments in apposition should it be necessary to use such. In these fractures the results obtained at present under any form of treatment are, in my experience, frequently most unsatisfactory. If on the dead body one breaks through the fibula in the position in which it is usually fractured, it is possible, by adducting the foot, to replace the fragments more or less imperfectly in apposition, and I should imagine that it was from the study of these fractures on the dissected body that the forms of splints used for their treatment were founded; but in the case of fracture in a living subject, where the injury is often followed at once by a certain amount of hæmorrhage, and after a very short

interval by considerable inflammatory effusion, I believe the forcible adduction of the foot not only does not serve to bring the fragments into apposition, but by tracting on the bruised and inflamed peronei, and upon the skin stretched by inflammatory exudation beneath it, the displacement of fragments is, if anything, exaggerated. In other words, I am of opinion that in a Potts' fracture in which there is a distinct displacement of the fibular fragments it is impossible to reconstitute that bone in its normal form by manipulation, while it may be effected with certainty through an incision a little more than an inch in length.

Passing to the consideration of fractures of the long bones of the upper extremity and of the clavicle, I would remind you again that these bones are more favourably placed than those of the lower extremity, since they do not transmit anything like the same amount of pressure through them. On this account a considerable amount of deformity or limitation of movement may ensue upon a fracture or fractures sustained during or after middle life, without their being accompanied, as a matter of course, by any marked progressive pain in the neighbouring joints. Yet in the arms mechanical changes of the "passive" type may develop occasionally to a considerable extent, and they are more marked and more depreciating to the value of the individual as a machine when the physiology, and consequently the anatomy, of the joints have been previously altered by the prolonged pursuit of a laborious occupation, such, for instance, as that of the coal trimmer or shoemaker. Therefore we may conclude that in fractures of the upper extremity it is not a matter of such vital importance to reconstitute the fractured bone or bones in their original form as it is in the leg.

In fractures of the clavicle, where there is much displacement which cannot be remedied by some appliance; and in young children, where great difficulty is experienced sometimes in preventing considerable overlapping ; and again in some comminuted fractures of this bone, the ends of the fragments should be exposed, drilled, and connected accurately with silver wire. The innumerable considerable deformities which are so frequently seen in consequence of fracture of the clavicle, occurring especially in young life, appeal strongly to the surgeon, and reproach him with incompetency, since he is satisfied to allow his hands to be tied by an obsolete creed rather than use his common sense in treating these conditions.

Again, in fractures of the humerus, involving either epiphysis, how much good can be done by exposing the fragments, placing them in accurate apposition, and connecting them, if necessary, by wire or screw, while under the ordinary methods a permanent limitation of movement and depreciation remains, or the subject is exposed to a subsequent operation, a joint being very probably resected.

I do not suppose that my experience is singular, but I have seen a large number of cases of fracture of the long bones of the upper extremity, in which there was considerable deformity, limitation of movement, loss of power, limited control over muscles, and occasional involvements of nerves, as the musculo-spiral, all of which would have been avoided by operative treatment.

It comes to this, that many of these conditions cannot be treated satisfactorily by methods other than operative, however skilful, able, or experienced the surgeon, while a surgeon possessed of a competent knowledge of surgery can deal satisfactorily with them by operation.

It has often puzzled me how a surgeon who does not hesitate to mutilate the bones of a rachitic subject with the osteotome, chisel, or saw, and who readily accepts the responsibility of excising an elbow joint which has become fixed from fracture of one of the bones forming it, should consider an operation on a simple fracture as of such a more serious nature.

Gentlemen, I do not wish you to go away with the idea that in every case of fracture of the long bones of the limbs in which you cannot place the fragments in accurate apposition, occurring in a subject dependent for his livelihood on the more or less perfect usefulness of that limb, or in one in whom symmetry of form is a matter of the greatest importance, you are to expose the fragments and to unite them with steel screws or wire any more than you should perform any other important surgical operation, unless you are confident, not only of your ability to do the operation skilfully, but also of the absolute cleanliness of yourself and your assistants, and that the surroundings of the patient as regards nursing, hygiene, etc., are such as would justify you in accepting what must always be a terrible responsibility, since failure in any of your precautions may entail on the patient loss of life or limb. If after careful consideration you are of opinion that an operation is the best course to pursue, and you

are convinced that you are justified in accepting the risk of its performance, you should inform the patient fully of the various results which are likely to ensue from mechanical arrangements and from operative measures, so that he may be in a position to decide as to what choice he will make. I think you will find that in proportion, as the surgeon acquires confidence in the safety of his operative procedures, begotten of a thorough knowledge, and careful application of the principles of aseptic surgery, so you will see him interfering actively with increasing frequency in the treatment of simple fractures of all kinds, and the various methods by which it is attempted to reconstitute fractured bones in their original form by means of splints, will, except in a small proportion of cases, fall into disuse. The treatment of fractures as it exists at present, is a disgrace to surgical practice.

A CLINICAL LECTURE

ON

MORBID CONDITIONS OF THE TYMPANIC ATTIC.

Delivered at St. Mary's Hospital, Mar. 7, 1895, by

WILLIAM HILL, B.Sc., M.D.,

Surgeon to Aural Out-Patients at the Hospital.

GENTLEMEN,—I propose calling your attention to-day to morbid conditions of that part of the tympanum which is situated above the ossicles and chorda tympani nerve, and which you have learnt in this department to associate with a perforated Shrapnell's membrane, a sign which is often, though by no means invariably present. We always have a small number of cases of attic disease under treatment here, and some five or six are now present for your inspection.

The specimens which I show will serve to recall to your minds, without detailed description, the position and anatomical relations of the attic. That part of the tympanum which lies immediately within the lower five-sixths of the drumhead—the membrana tensa—may be designated the tympanum propium or atrium, and it is more or less shut off by the ossicles and ligamentous and mucous bands from the space above, and this upper space is, therefore, well named from its position, the tympanic attic. Politzer speaks of this region as the cupola space; some otologists

have designated it the malleo-incudal recess, and others, again, the epi-tympanic space, and the last is, perhaps, the preferable term; but as the majority of aural surgeons in this country have adopted the short word attic, we need not trouble about preferential nomenclature.

Please observe that the attic is limited above, and cut off from, the cranial cavity by the thin layer of bone known as the tegmen tympani, which sometimes, especially in young subjects, is further weakened by an unobliterated petrosquamosal fissure; internally, the attic is bounded by that portion of the petro-mastoid which is adjacent to the pyramid and to the horizontal part of the aqueduct of Fallopius above the foramen ovale; posteriorly, is seen the aditus leading to the so-called mastoid antrum. I say so-called because this antral cavity is bounded by the squamous as well as by the petro-mastoid element of the temporal bone, and I have suggested elsewhere that the term temporal antrum is less open to objection. Externally, the attic is limited above by the squamosal bone with its cell spaces, and below by Shrapnell's membrane, which extends between the pars ossea or Rivinian segment of the squamosal down to the crescentic anterior and posterior folds of the malleus. In an anatomical section made coronally through the attic you will observe that, though it communicates normally pretty freely below with the tympanic cavity proper in front and behind the chain of ossicles, yet such structures as the tensor tympani muscles, the stapedius muscle, and the chorda tympani nerve further tend to fill up the floor of the attic; and it is important to remember this because, in tympanic inflammations, adhesions and membranous bands form which are sometimes so extensive as to cut off partially or entirely the tympanum proper from the attic. Moreover, the attic itself is traversed by the superior and external ligaments of the malleus which subdivide the cavity into an external and internal attic, and this has to be borne in mind, because we meet with cases in which the external attic, as the result of middle ear inflammation, gets completely shut off and isolated by bands and adhesions from the internal attic, whilst the latter may, and often does, escape serious damage—that is to say, as long as its communications with the antrum above and the tympanum proprium below are not materially obstructed. It is probable that most cases of obstinate attic disease commence in the external attic (Prussak's space), which, as you

will see, is immediately in relation with the external ligament of the malleus, the head and neck of the malleus itself, and with Shrapnell's membrane.

In the cases here to-day you will only be able to observe, ocularly, from the point of view of the aural speculum, disease in the outer wall of the attic ; in some of the patients you will only *see* a hole in Shrapnell's membrane, whilst in others, in addition to destruction of this structure, the hole is observed to be further enlarged at the expense of the squamosal bone in the neighbourhood of the Rivinian segment, and in one case at least, the squamosal cells are exposed by the carious process. Most of these patients have had either polypi or cholesteatomatous masses removed from the attic, or granulations appropriately treated through the Shrapnell's perforation : in one the incus came away through the perforation ; certainly in two instances the attic probe demonstrated caries of the head of the malleus, and there is a patient in the Albert Ward, under Sir William Broadbent, whose tegmen is probably destroyed, for a probe when passed through Shrapnell's perforation apparently enters the cranial cavity, and he has unequivocal symptoms of cerebral abscess. You will see, therefore, how necessary it is to have an accurate conception of the anatomical relations of this important region.

Before demonstrating to you these lesions in the persons of the patients shown here to-day it will be convenient for me to give you a short systematic account of the more practical points in connection with this important class of cases.

Acute inflammation limited altogether to the attic must, I think, be comparatively rare, though this space doubtless usually participates in most acute catarrhs of the middle ear as a whole ; but our attention is not particularly drawn to the area, because it is the highest point of the entire tympanic cavity, and the bulging of the drumhead, due to excessive accumulation of fluid and subsequent rupture, is seen at a more dependent point, viz., in the postero-inferior segment of the membrane. During the recent epidemic of influenza, however, some of you will remember a patient seeking advice in this department whose ear-ache had been relieved the same morning by the drumhead bursting, and on inspection we found a sero-sanguineous fluid issuing from a well-marked Shrapnell's perforation, the rest of the membrane appearing a little red, but otherwise normal. Such instances of acute inflammation are uncommon.

On the other hand, chronic lesions in the attic, with or without acute exacerbations, and associated or not with perforative disease of the membrana tensa, whilst not coming under observation every day, or even every week, are sufficiently numerous to enable me to show you no less than six on the present occasion, and I have treated a considerable number of cases of active attic disease within the last seven years. Small dry perforations of Shrapnell's membrane without active disease are, I need scarcely say, often met with.

As to the etiology of the condition, I can only state that there is nearly always a history of a former acute median otitis, the causes of which I discussed at length some weeks ago. Mr. Field,* however, says, " morbid changes develop in this region partly as *primary* affections and partly as the result of diffuse diseases of the tympanum." If the inflammatory products of acute middle ear disease are not speedily absorbed either from want of appropriate treatment, or from the formation of bands and adhesions with retention of secretion, or from the brunt of the attack—as in the virulent otitis sometimes resulting from the acute specific fevers—having fallen on the attic, then this part of the middle ear may be the seat of ulceration, of granulations, of polypi, or of cholesteatomata ; and these lesions are usually associated with perforation of Shrapnell's membrane—Nature's attempt to cure by drainage—and often with caries and necrosis of the squamosal bone in the neighbourhood of the Rivinian segment. In some instances, there is evident disease of the ossicles, and occasionally erosion of the tegmen attici follows. In the ear, as in the nose, the appearance of granulations (not following traumatism or operative procedures) will at once raise the suspicion of bone mischief, which is generally verified by the cautious use of the probe. Whilst granulations and polypi are usually the result of caries, cholesteatomatous masses, on the other hand, which increase in size by surface accumulations, seem, by reason of this increase, to bring about absorption of bone by pressure ; and it is in this way that the cranial cavity, the squamous cells, and even the antrum are in some instances invaded. The *débris* removed from a diseased attic has been proved over and over again to be frequently the habitat of numerous pathogenic microorganisms, including the tubercle bacillus and various septic cocci, which are often present ; and of course, the less dangerous fœtor-producing

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" Diseases of the Ear," 4th edition, p. 221.

microbes can be demonstrated whenever the discharge is foul smelling. Cholesteatomatous masses usually stink, and granulations may do so; but granulations removed from the attic, which have no appreciable odour, often teem with pathogenic organisms, which render them far more likely to lead to dangerous complications than, for instance, cholesteatomata, when the latter are infected only by fœtor-producing microbes. The point to be insisted on is, Do not be led to think lightly of a case because fœtor is absent, and hesitate to consider a patient necessarily relieved because deodorizing instillations have reduced the stink from the ear to a minimum. I shall return to this subject when the question of treatment is under discussion.

I now pass on to the question of symptoms. More or less deafness is the rule, though I have notes of exceptions to it. Some patients seek advice on account of discharge from the ear and deafness; some for discharge, together with pain in the ear or head; some for pain or headache alone; others, again, complain that the discharge, usually scanty, has suddenly ceased, and that they have suffered from otalgia or from headache, and increased deafness ever since; occasionally one is consulted on account of hæmorrhage, which is sometimes quite profuse, the patient giving a history of having suffered for a long time from a discharge and deafness, with intermittent pain. Tinnitus is present in many cases, but vertigo, though occasionally severe, is much less frequent. The disease is insidious, and there is usually a history of an acute otitis, followed by a chronic and perhaps intermittent discharge, extending over an extended period, which is more often reckoned in years than months. Discharges from the attic itself are rarely profuse, but, inasmuch as a number of cases have concomitant suppurative disease of other portions of the middle ear, only uncomplicated cases are typical in this respect. The discharge being thick and scanty, the intermittency of the flow from the attic, the blocking up of the perforation with *débris*, and the subsequent drying of the same to form a crust, explain the onset of pain and headache, and the increase in deafness and other symptoms consequent on the damming back and retention of septic accumulations in the attic. You all know the traditional objection which existed in the minds of members of our profession a generation or so ago, to the adoption of measures to cure an aural discharge, and the objection is still shared by a section of the

laity, and it was, no doubt, founded on the evil results observed to follow a stoppage, without a cure of the discharge; and the fact that when later the dammed back secretion again bursts forth immediate relief is afforded naturally appears to the ignorant as a confirmation of this heterodoxy. There is, however, all the difference between stopping and stopping up a discharge.

In reference to pain, it is not complained of in all cases; if present, as often as not it is referred to some part of the head rather than to the middle ear or mastoid. General headache is not uncommon, but pain in the temple or in the supra-orbital region is, I think, more usual, though it may be located at the vertex or at the occiput. When there is aural pain there is frequently headache as well. If attic disease leads to morbid complications within the cranial cavity, the aural symptoms are of course masked by the graver ones severally associated with brain abscess, meningeal inflammations, and abscesses, or pyæmia, but this important aspect of the subject it would be inconvenient to discuss now; and I will not digress further than to impress on you the fact that a certain proportion of patients with attic disease, and especially those which are untreated, eventually suffer from intracranial complications, which are always dangerous and frequently fatal. The first symptom of this, which often long precedes all others, if we except headache, is mental hebetude and a sleepy, apathetic condition. It is by no means diagnostic of intra-cranial extension, being sometimes present in uncomplicated attic disease, but if unrelieved after appropriate aural treatment the gravity of the case is immensely increased. Attic disease is not uncommonly associated with disease in the contiguous antrum and mastoid cells, and this combination of morbid lesions will be further alluded to when we come to consider Stacke's operation, for exposing the entire diseased area, under the head of treatment.

I need scarcely add that sooner or later the general health suffers, and that exceptionally even visceral sequelæ follow, especially when there are tubercular granulations in the attic.

The objective appearances, as observed with the aural speculum, are very varied; to simplify the discussion of them I will first of all direct your attention to the more obvious cases of chronic attic disease in which there is a perforation in Shrapnell's membrane, and later I shall discuss those in which there is no Shrapnell's perforation,

but a hole or holes in the membrana tensa, though with attic lesion, which can be inferred only from a consideration of the course of symptoms and from information gained by the intra-tympanic syringe and probe.

When the external attic is involved, either with or without lesion in the internal attic, there is probably always a perforation in Shrapnell's membrane ; if it is not obvious on examination with the speculum, it is either from the formation of a crust due to the drying of the scanty thick discharge over the hole, or the perforation may be obscured by the presence of granulation polypi, or by suppurative discharges. The meatus, and especially the inner end of the meatus, must be thoroughly cleansed by appropriate means, therefore, before the presence or not of Shrapnell's perforation can be decided on ; it is necessary to tilt the head of the patient well over to the opposite side in examination with the speculum, as Shrapnell's membrane is often situated nearly on the same plane as the roof of the meatus. A very small *dry* perforation, situated just above the short process of the malleus, about the size of a pin's point, is frequently seen in quite healthy ears ; it is no indication of even former disease, and the same applies to the presence of a very small pit in this position, which the novice might mistake for a perforation ; a large *dry* pit, or even hole, in Shrapnell's membrane, with no headache, and no discharge from any part of the middle ear, indicates former but healed attic disease. On the other hand, holes in this part of the membrane, of whatever size, whether large or small, which are *moist*, usually point to active attic disease, and must be regarded with the greatest suspicion. If the perforation is not covered by a crust it will be seen to be more or less blocked by thick pus, by a cholesteatomatous mass, or by a granulation or small polypus ; very exceptionally, a sequestrum, such as a carious incus, the head of the malleus, or a detached portion of the bony boundary of the attic, may be found blocking the hole. Concomitant perforation and scarring of the membrana tensa is not infrequent—probably the rule. In very large attic perforations there will be evident loss of substance of the Rivinian segment of the squamosal bone, as is exemplified by a man now attending here. You will remember that Dr. J. L. Morton removed by avulsion a large polypus, the thin pedicle of which passed through the perforation into a carious attic. The man's headache has

disappeared, and his hearing has improved since the attic has been efficiently cleansed and drained.

Having discovered an attic perforation and cleansed the membrane of pus, crusts, or *débris*, the next diagnostic procedure is to ascertain the condition of the attic by the gentle use of special straight and angular probes inserted through the perforation. Ossicular disease, if present, can thus often be made out with the straight probe directed inwards ; the attic walls can be explored, and much information as to the dimensions and contents of the cavity gained by gently rotating small angular probes. Free bleeding raises a strong suspicion of granulations, and the difficulty experienced in rotating even a small angular probe from the resistance of a doughy material points to blockage of the cavity by a caseous or cholesteatomatous mass. The probe can often be advantageously preceded by the use of the intra-tympanic syringe. When there is a suspicion that the tegmen is eroded in addition to the angular probe a curved one is useful, but this form of deep probing into the cranial cavity, though occasionally indicated, may be fraught with much mischief, and should only be undertaken by an expert. A small spoon or scoop passed into the attic will sometimes bring away portions of cholesteatomatous material, and so clear up the diagnosis. A word of warning, however, is necessary in reference to the use of the probe, curette, and scoop for this purpose, and I cannot do better than quote you a paragraph from the Harveian Lectures of 1892, by Mr. Field :—"Such procedures may awaken the latent energies of septic micro-organisms which abound in them, and niggling operations for their removal have more than once resulted in sharp attacks of otitis, accompanied by high fever and threatenings of meningitis." These manipulations usually cause pain, and the hearing power may be worse afterwards, or, on the other hand, it may be immensely improved. Intra-tympanic syringing causes more or less vertigo, which however usually passes off in a few minutes, and rarely lasts more than half an hour.

The diagnosis of attic disease when there is no beacon, like Shrapnell's perforation, to guide us is a more difficult matter ; it should, however, be suspected, in those cases of long-standing otorrhœa, where, with occasional headache in the temporal or frontal regions and with no obvious granulations present, and in spite of appropriate treatment, thick pus is seen, through the perforation in the mem-

brana tensa, to come from the upper and fore part of the tympanum, and especially must it be suspected in those cases where the headache, pain, and deafness is obviously increased when the discharge suddenly ceases to make its appearance in the meatus. In these cases it may reasonably be suspected that the ossicles and other structures crossing the tympanum, together with adventitious bands, granulations and caseous masses have helped to dam back the thick discharge, and the diagnosis lies between attic lesions and antral disease; probably the two conditions are often co-existent. In the absence of positive symptoms pointing to serious involvement of the antrum and mastoid cells, we can safely assume that, even if these areas are involved that the primary mischief is in the attic, and the intra-tympanic syringe, with an angular nozzle and the angular probe, inserted through the perforation, may be used to clear up the diagnosis in the same careful manner as was recommended in exploring those more obvious cases with Shrapnell's perforation. Hidden granulations readily bleed when touched with the angular probe, bone disease and cholesteatomatous masses can often be felt by it, and the intra-tympanic syringe may succeed in dislodging caseous *débris*.

Coming now to the question of treatment, it must be admitted that lotions syringed into the meatus in the ordinary way rarely reach the attic, and generally therefore are by themselves useless; powders and drums are actually harmful in this condition. Before adopting more radical measures, syringing out the attic once—or better still, twice —daily, with solutions of peroxide of hydrogen, sanitas, biniodide of mercury, and other antiseptics, by means of the intra-tympanic syringe ought, where practicable, to be tried; it is, however, obvious that this treatment cannot be applied to out-patients in hospital practice. Should intra-tympanic syringing fail to relieve, or be impracticable, the question of thoroughly curetting the attic with scoop or ring knife, and applying chromic acid to granulations, if they be present, has to be considered. This treatment is rational enough, and if thoroughly done, often succeeds; but an anæsthetic is usually required, as few patients can stand this painful procedure under cocaine alone, and moreover the patient must be put to bed afterwards and prepared to stay there for some days, as marked vertigo and prostration, together with pain, often last a week or two, but these eventually subside. There are certain

dangers connected with this apparently simple procedure; the ossicles may be injured and dislocated, and the hearing thereby affected; septic organisms may be stirred into activity, and grave inflammation set up in the temporal bone, necessitating the opening of the mastoid; and lastly, temporary facial paralysis may ensue.

In two cases in which I curetted the attic for granulations through a Shrapnell's perforation the most alarming prostration and vertigo ensued, and the patients were in a very tottering condition for some weeks—one whom I saw with Dr. Tweed was a lady of 35, whose perforation in Shrapnell's membrane quite healed, there is no trace of granulations or discharge now, and strange to say her hearing is perfect.

If these methods fail to relieve the symptoms, it is probably because of the presence of cholesteatomata, or the ossicles are diseased, or else adventitious bands attached to the ossicles prevent efficient drainage from the internal attic and antrum; in these cases it is no doubt good surgery to remove the obstructing malleus and, if necessary, the incus, to enable us to expose and thoroughly treat the diseased area. The operation (which is specially indicated when there is disease of the internal attic) though delicate, is not a difficult one, it has been frequently performed abroad under cocaine, but a general anæsthetic is unusually administered in this country; it is liable to be followed by the same alarming symptoms that often ensue after simple curetting, and whilst the hearing power is rarely improved it is occasionally made worse. Still, the results are often astonishing. In the case of a nurse sent to me here by Mr. Steer some three years ago, the pain and discharge due to attic disease was completely cured by removing the malleus and curetting under an anæsthetic.

If removal of the ossicles fails to relieve, we may generally be sure that the case is complicated by granulations or other morbid lesions in the antrum or mastoid cells, and much more radical measures may be required. In Stacke's operation the auricle is displaced forward, the antrum opened, and the posterior wall of the meatus chiselled away so that the antrum and attic are exposed as a continuous cavity, when their morbid products can be removed. I shall have more to say about these more serious cases and the varying results of treatment on a future occasion; and time prevents me speaking on the technique of the intra-tympanic operations which I have advocated.

THE CLINICAL JOURNAL.

ON THE SURGICAL TREATMENT OF EMPYEMA.

A Paper read before the South Eastern Branch of the British Medical Association at Dorking

By JOHN H. MORGAN, F.R.C.S.,

Surgeon to Charing Cross Hospital.

WHEN we reflect upon the strides and advancements which surgery has made during the past quarter of a century; when we see the abdomen opened daily for the removal of growths affecting every organ contained within its cavity, often of the largest size, and attached to the most important viscera; when we see it opened and explored with impunity for diagnostic purposes only; when large collections of pus are removed, and important viscera, and even sections of intestine successfully ablated; when we see the skull trephined, and abscesses of the brain successfully opened and drained; and when we see tumours of the cerebrum removed and perfect healing taking place after all these and many other operations, it is fair to inquire whether in other cavities of the body, the progress of surgery has advanced with equal strides.

If we take into consideration the surgery of the thorax, it is true that the pericardium has of late been frequently tapped and even incised for the evacuation of large quantities of fluid, and many cases have recovered completely who would probably have died if the operation had not been performed.

Cavities of the lung due to bronchiectasis, gangrene, or hydatids have been opened and drained with success, and to both these operations there are still many possible future developments.

But with regard to empyema, it is interesting and important to inquire whether surgical proceedings in regard to this affection have kept pace with these developments in other parts of the body.

A recent writer, Mr. Godlee, who has had large and exceptional experience at the Brompton Hospital, concludes an article on this subject by expressing the belief that although great advance has been made in recent years, we have not yet arrived at the best possible method of dealing with old chronic cases of empyema.

In order better to appreciate some of these advances, let us turn for a moment to the work of Sir Thomas Watson, whose writing is always of the greatest charm and interest. After quoting two cases which had come to his knowledge, in which paracentesis thoracis had been determined on to relieve the oppression caused by empyema, but in both of which the opening was made on the wrong side, and in each case death had ensued in three minutes, he says that both of these cases occurred some years ago. "Such a mistake would be unpardonable now." He then relates a case in which a trochar was thrust into the chest, but no fluid followed, to the no small mortification of the physician. This proved to be a case of malignant disease of the lung, and fluid was let out afterwards by puncturing the thorax in another place, and much relief afforded, although, of course, the disease proved ultimately fatal. If we follow his directions we cannot, even at the present day—and these words were published fifty years ago—have clearer directions as to the indications for using the trochar.

"You will take care, then, to survey the chest narrowly before you plunge a trochar into it. If you see by your eye and ascertain by measurement that one side is larger than the other, if the intercostal depressions be effaced on that side, if the whole surface affords a dull sound when percussed, if the side does not move at all, or scarcely moves during respiration, if no vibration can be felt on that side when the patient speaks, if no breathing can be heard in the corresponding lung, if the heart be found beating in an unnatural place down towards the left hypochondrium or in the other direction on the right of the sternum, and if, at the same time, the other side of the chest moves freely, sounds resonantly, communicates a thrill to the hand while the patient converses and is full of puerile respiration, then you may be sure that the larger side is distended with fluid."

Some of his further injunctions would no doubt be modified at the present day when the risks of opening the thorax are so much diminished, but his

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final dictum remains a law—viz., " whenever (no matter how we ascertain the fact) the effused liquid consists of pus it should be let out."

Can we at the present day state the case more clearly, and have we any means by which our diagnosis can be made more clear or our course of action better determined?

It is true that the science of auscultation and percussion is more carefully studied and taught, and the mistakes against which he cautions us would more than ever be unpardonable.

In addition to the means at his disposal beside the stethoscope, we have the clinical thermometer, but this, in such cases as we are considering, is often not reliable. Pyrexia and hectic may be absent or little marked in empyema, especially in long-standing cases, the alteration of the pleura or the degree of tension preventing absorption. Occasionally the disease is latent many months. Well-marked pyrexia may be present in serous effusions; thus in these the evening temperature may reach 101°. (Jacobson).

Watson mentions the desirability of establishing the diagnosis by the insertion of a grooved needle, but this instrument is now replaced by the aspirating needle and syringe. With the aid of this instrument the presence of pus can be much more accurately ascertained.

And now, having ascertained the presence of pus, what are the means to hand by which it can be evacuated? Formerly the trochar was the only available means to this end, but since the advent of antiseptic surgery its use has been almost entirely abandoned, and so has also the method of subaqueous drainage, which came, as it were, midway between the use of the open trochar and the aspirator. We have then the choice of three methods :—

1. The aspirator.
2. Free incision.
3. The removal of a portion of rib.

The aspirator is not to be despised as a temporary measure and, no doubt, is often productive of cure, after a single, or even after repeated applications. This is more likely to be the case when the effusion is semipurulent or very localised. It is adapted especially to such cases and also to very young children under 2 years of age, in whom the results of excision of rib are not satisfactory. But in older patients the use of this apparatus is very often deceptive and even prejudicial to the patient. When an empyema is

tapped by this method a certain amount of relief is afforded, the breathing is easier, the temperature falls, and the condition of the patient for a while improves. But, as a rule, the symptoms gradually reappear, the cavity again becomes filled, and the same operation is repeated with probably less favourable result. Meanwhile, the adhesions of the pleura are more confirmed, the compression of the lung continues, and its power of expansion is lessened, and the strength of the patient wanes.

As a means of cure then, the aspirator may only be relied upon in the case of very young patients, or in cases where the collection of pus is very small and strictly limited in area, whilst in older patients it may be used as a means of temporary relief pending more radical measures.

Of the other two methods, it is generally admitted that the resection of a portion of the rib is far the most satisfactory operation in the case of children. Firstly, because the close approximation of the ribs does not allow of exploration by the finger, and secondly, for the same reason and by their movements upon one another the drainage tube is liable to be compressed, and the free issue of pus through it is liable to be considerably checked.

The operation is simple, easy, and quickly performed, and by the opening so made a free exit is given to all contained matters, and the extent and ramifications of the cavity are easily explored, and adhesions which may be the means of confining smaller collections can be easily broken down.

As to the position in which this opening should be made, of course in the case of a localised empyema the surgeon has no choice but to make his opening as near as possible to the lowest part of the cavity. But where the whole of the pleural cavity is presumed to contain a purulent collection some differences of opinion exist as regards the most favourable situation. My own preference in the case of children is for the seventh or eighth interspace somewhat behind the mid-axillary line, but in adults the ninth interspace may often be chosen with advantage.

The patient being under the influence of chloroform—which is the best anæsthetic agent in such cases—and all antiseptic precautions having been taken, the arm on the affected side is raised to a right angle, and a free incision carried down to the upper border of the selected rib. The periosteum having been raised from the front and back part of the rib for at least an inch and a half,

the bone is divided at each end of this space by strong cutting pliers, and when the bleeding, which is seldom troublesome, has been arrested, a steel director is thrust through the inner layer of periosteum and on through the thickened pleura. The pus should at once issue, and the opening is then enlarged by means of a pair of dressing forceps, which should be opened widely. As soon as the greater amount of pus has flowed out, the finger is introduced and the necessary exploration made, by which adhesions are broken down, and the position and amount of compression of the lung is ascertained.

By the finger also much advantage is gained in the loosening and removal of large flakes of lymph which, if left, form foci of septic organisms. Much hesitation is generally felt by surgeons about washing out the cavity thus opened, and cases have occurred in which alarming, and even fatal consequences, have followed this proceeding. I have, however, adopted it on several occasions, and have never seen any bad result, and I believe that where the contents are foul or the lymph abundant it is indicated. I have used warm boiled water, followed, if necessary, by boracic lotion.

The drainage tube should at first be large and long enough to enter the pleural cavity, but may be made shorter and smaller after the first or second dressing. The end that protrudes through the skin must be secured to a piece of flat india-rubber or to some bunches of catgut, in order to ensure against its slipping into the thorax, an accident which may easily occur unless good precaution is taken. Moreover, in removing the dressings it is well to be sure that the drainage tube has not come away with them, as if it should be missed when the dressings have been thrown away, a great deal of anxiety may be groundlessly raised. Should the accident unfortunately occur the searching for the tube is a very difficult and often tedious process. It has happened on two or three occasions that children with open sinuses have been brought to the hospital with the story that a drainage-tube had slipped in after the pleural cavity had been opened, and the search for it has always been a most anxious and prolonged task. I do not think that any rule can be laid down as to the time at which the drainage tube should be removed. This must, of necessity, vary in individual cases, and must depend upon the amount and character of the discharge, but it

may often be removed with safety on the third day and generally by the tenth. It is probable that when the tension of the empyema is released, and the pus is evacuated, the natural absorbent powers of the pleura are restored, and the seropurulent secretion from the walls of the diminishing cavity is very largely re-absorbed.

The dressings applied over the wound should be of a strictly antiseptic character and should extend over such an area as to prevent the possibility of the discharges becoming fœtid by contact with the air. The discharges which ensue after the first opening of an empyema are very profuse and voluminous even when every care is taken to empty the cavity, and this may require that the dressings should be renewed after a few hours, but the period may be prolonged if the dressings are sufficiently ample to soak up and absorb all the fluids discharged through the tube, and after the first two or three dressings, even if the drainage tube is not removed, the discharge will generally dwindle to very small amount.

It must be remembered that the dressings, besides forming an antiseptic filter, act also as a valve to prevent the access of air to the cavity, as the chest resumes its inspiratory action and thus forms a check to the putrefactive changes which would otherwise be so prone to occur.

It is the practice of some surgeons to use incision in the case of children, but, as I have already pointed out, the advantages of removal of a piece of rib are so great that it has become an almost universal practice at the Hospital for Sick Children. In adults the case is different. The space between any of the first six interspaces is broad enough to admit the index-finger, and therefore by this means all is gained by an incision which can only be afforded in smaller patients by the more extended operation. Nevertheless, I am personally of opinion that in adults the more extensive operation gives the better results.

In the year 1893, between the months of February and July, I performed this operation upon eight patients. The empyema in six was on the left, in two on the right side. Three of the patients were children under 10 years of age, one was under 17, and four were over 30.

All these patients recovered perfectly except one, who came into the hospital with a sinus in the left side, being the result of an incision which had been made by another surgeon before his admission to Charing Cross Hospital. I removed a

piece of rib, and his condition greatly improved, though the discharge continued to a small amount when he was obliged to leave the hospital on account of returning to his business.

No doubt the power of the lung to expand is far greater in children than in adults, and it is probable that the process of repair is somewhat different in the two cases, depending in the case of children mainly on the re-expansion of the lung and less rigid condition of the chest wall, which allows it to fall in, and in the case of adults on the gradual contraction of organizing granulations which draw the parts together, and the raising of the diaphragm. Thus the success of the operation is more frequent in the case of children than in adults, and the time requisite for the lung to regain its proper functional state is necessarily much shorter in the case of the young than in that of the adult.

There seldom occur any drawbacks to the satisfactory progress of the patient when once the confined matter has been given a free exit. It may, however, happen occasionally that some of the discharge becomes pent up, probably behind some pleural adhesions which have not been broken down, and this is soon evidenced by the temperature chart and the general condition of the patient. This happened two years ago to a young gentleman, aged 10, for whom I opened a large empyema on the right chest, at a private school in this neighbourhood. A very large quantity of pus was evacuated, and this was of so foul a character that I washed out the cavity with a weak solution of perchloride of mercury, and directed that it should be syringed out daily with a weak lotion of boracic acid. All went well for three weeks, when I was summoned by telegram to find that the temperature had suddenly risen to a very high point, and that hectic had again supervened, and that, in short, there was either matter confined in some part of the cavity, or that a fresh abscess was forming. I found the wound looking perfectly satisfactory, and that the re-expansion of the lung had gone on to a large extent. On passing a long probe upwards towards the clavicle my anticipations were satisfactorily realised by a gush of pus which had evidently accumulated behind some inter-pleural adhesions, and the case subsequently went on without drawback to perfect recovery. The operation was performed on March 9th, and I heard from Mr. Walker, under whose care the boy came on his return home, that before the end of the year he was successful in winning one of his school races.

Satisfactory as are the results of these operations, it must not be presumed that every case of empyema is to be subjected to either one or other method. In chronic phthisis the question is one of very serious consideration, and the opening of the chest may do no harm, but may be followed by very serious constitutional disturbance. Again, when there is evidence that an empyema has made an opening into a bronchus, it is probably better left to the course of nature. The same may be said of those cases in which the matter finds its way, as very rarely happens, into the stomach or some portion of the intestines.

It was formerly considered that radical operation was forbidden in the case of double empyema, and that whilst the collection on the one side might be treated by one of the measures which have been discussed, aspiration was the only means of dealing with the other. But, in 1890 I published the notes of a case of a boy aged 6, who was admitted into hospital with fluid on both sides of the chest, more extensive on the left, where there existed dulness everywhere below the second intercostal space. The apex of the heart was pushed downwards and inwards, and was situated below the margin, and a little to the left of the xiphoid cartilage. On the right side, dulness extended from the upper third of the vertebral border of the scapula to the twelfth rib. He was relieved by the withdrawal of eight ounces of fluid from the left chest, and later from the right of 22 ounces. The boy was rapidly losing flesh when I was called in, and by resection of a piece of rib from the left side a very large quantity of healthy pus was evacuated; great benefit ensued at once, but the condition of the right side prevented him from making any very great improvement. A week later, twelve ounces were removed by aspiration from the right side; but on the twelfth day after the first operation a similar proceeding was followed on the right side, when a very large quantity of pus came away, so large indeed, that it was not thought advisable to turn the child on his side lest the evacuation should be too rapid. The boy made a complete and speedy recovery, and was discharged with both openings soundly healed, and was seen a month later in perfect health. He was inspected in June, 1890, exactly a year after his discharge, and it was found that the periosteum in the region, on both sides from which portions of rib had been removed,

had thrown out bone which formed a firm covering to that part of the thorax. Respiration over the whole chest, including the situation of the empyemata appeared perfectly normal, and the heart's apex was in its natural position.

It may be gathered from what has been said that I regard the early opening of an empyema, with the exceptions that have just been mentioned, as of imperative importance. So soon as the presence of pus has been surmised by the usual symptoms, the aspirating needle should be introduced, and if pus be withdrawn a freer exit must be given by one or other of the operations advocated. Not only will this give the lung a better chance to expand and to regain its proper functional state, but by diminishing the amount of chest contraction it will tend to avoid that tendency to curvature of the spine which so often results from former pleurisy or empyema. My colleague, Mr. Pitts, in his lectures at the College of Surgeons, relates that he inspected 26 patients who had been operated upon at a former period at the Hospital for Sick Children. No resulting deformity was perceptible in the majority—22—of these cases, and in the remaining 4 it was very slight. At the same time the possibility of deformity must not be overlooked, and as soon as health will permit every effort must be made to restore the full and normal action of the diaphragm and other respiratory muscles, which become weakened on the affected side, partly by inaction, and partly by a certain amount of inflammation of the muscular tissue. To this end artificial respiration should be resorted to two or three times a day, and later all such exercises as will tend to expand the chest—carefully regulated gymnastics, drilling, etc.—should be carried out over a considerable period of time.

I believe that the recognition of the advantages of early operation are gradually reducing the mortality as well as the ill effects of empyema. Mr. Pitts, in the lectures already mentioned, found that between the years 1880-92 there were treated at the Hospital for Sick Children 83 cases in which an incision was made. Of these 51 recovered, 16 were relieved, and 16 died.

During the same period of twelve years, 129 cases were treated by excision of a portion of rib, 99 recovered, 7 were relieved, and 23 died. On the other hand, in the report of the same hospital for 1893, I find that 50 cases of empyema were admitted, of which 2 died, neither having been operated upon; 42 operations were performed, resection of the rib being done in all of them, 41 recovered, and only one was reported as relieved.

I do not propose to discuss the many varieties and causes of empyema, but it seems to me that a consideration of its treatment is more than ever of importance at the present time, especially as during the last four or five years there can be little doubt that the recurrence of epidemics of influenza tends to the multiplication of cases of pneumonia, which so frequently involves the pleura in inflammation that results in the collection of pus within its cavity. It has been observed at the Hospital for Sick Children that cases of empyema occur in seasons or cycles during certain periods of the year. Whilst in some months there are no cases of this disease admitted, at others they will accumulate in large numbers. The explanation of this fact I must ask others to afford, but at the same time the subject of the treatment of the affection is one which affords the greatest interest, and deserves the most careful consideration.

NOTES OF CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Tuesday, March 26, 1895.

Feeble Circulation, with Chilblains and Œdema.

The subject of this case was a girl, aged 18, well grown, but of flabby tissues. Her hands were puffy and swollen and of a dusky red tint, spotted over with blotches of a salmon colour like those on the skin of a plaice. She had many excoriations, which were, doubtless, of the nature of chilblains. Her pulse was exceedingly soft, feeble, and compressible. The most interesting part of the demonstration, however, concerned the legs. Up to the tops of her boots the feet and ankles were not in the least swollen, and the skin was quite pale and natural. Above the boot and up to the knee the leg was swollen and dusky with congestion. A thick ring of solid œdema bulged

over the boot top. The girl had been engaged in household work during the recent cold weather, but had not been subjected to any hardships. Her mother, who came with her, showed her own hands in almost precisely the same condition.

Comments. It is of great importance that we should patiently study the simple and common courses of local disease. In investigating the phenomena of Bazin's malady we have often had occasion to remark that the pathological processes occur only between the ankle and the knee, and I have repeatedly taken occasion to remark that the absence of the disease on the feet is to be explained by the way in which the latter are protected. The leg, especially in women, is a part which is peculiarly exposed, and becomes, on this account, liable to peculiar diseases. It is, in common with the whole of the lower extremity, at a disadvantage as regards its circulation, and liable to venous congestion. In the foot the effects of this are wholly prevented by the tightly-laced boot, which is usually as small as can possibly be worn. The effects of the retarded circulation become apparent consequently above the boot-top, and many young women go about with a condition of slight solid œdema of this part of the leg. It is not at all uncommon to see a definite ring where the boot touches, and to find the subcutaneous cellular tissue thickened and solid above. No better demonstration of this state could be offered than in the patient before us. The girl is not the subject of dropsy nor probably of any organic disease. She has simply a very feeble circulation, and she has been obliged to stand and walk all day long without having any adequate protection to her legs. Hence the local conditions which we have seen. If she were the subject of inherited scrofula she would probably have inflammation of some subcutaneous lymphatic trunks, which would develop the nodules which precede the ulcers of Bazin's malady. The methods of treatment are obvious. She should rest more, take tonics, and give good mechanical support to the legs. Under the latter head a Martin's bandage would be the best expedient.

Keratitis and Syphilitic Teeth.

Mr. Waren Tay has been good enough to send us this boy in order to demonstrate the syphilitic physiognomy and teeth. They are very characteristic. He is just recovering from an attack of keratitis, or, I might say, kerato-cyclitis. He has conspicuous scars at the angles of the mouth, and his frontal eminences are unduly prominent. The first glance at his face would induce any skilled observer to suspect inherited syphilis. On looking at his teeth we find the upper central incisors notched and dwarfed, whilst the lower ones are rounded and peg-like. I do not attach any great importance to the latter condition, but his upper central incisors are as good a pair of syphilitic teeth as you could possibly see. We can form a very good opinion as to whether he took mercury in his infancy by inspecting his first permanent molars. They are the test teeth for the stomatitis which usually results from a mercurial course. On looking into his mouth I find his pre-molars well covered with sound white enamel, whilst every one of his four first molars are destitute of enamel on their surface, and show a roughened and craggy condition of the exposed dentine. There can, therefore, be no doubt that he did suffer from stomatitis during early infancy, at the period when these teeth were undergoing calcification. As he is the subject of inherited taint it is in a high degree probable that the stomatitis was from mercury.

Extensive Destruction of the Bones of the Palate and Nose in association with Lupus.

The subject of this case was a young woman, aged 26, who was brought by Dr. Thompson from the Moorfields hospital, where she was under the care of Mr. Morton. Her nose had been destroyed level with her face. The whole of the soft palate had also been destroyed, and its sides were united posteriorly to the pharynx. The hard palate and the middle of the alveolus had also been lost, and there remained only a bridge of gum under her upper lip. In addition to this very extensive destruction, the patient had a number of characteristic patches of apple-jelly lupus on the face, and a large one on the right forearm. Her left ear had been extensively destroyed by ulceration which had left edges as abrupt as if the tissue had been cut away with a knife. The scar about the ear and those in the mouth and in connection with the nose were perfectly sound, not the slightest trace of any lupus tissue being present. The scar in the right cheek was deep and adherent to the malar bone. The patient was absolutely deaf, and had suffered from keratitis in

both eyes. She had also a bad cough and a phthisical aspect. Her mother, who came with her, stated that several relatives had died in consumption, but gave no history which supported the diagnosis of syphilis. The patient was one of a family of several children, some older and some younger, all of whom were healthy. As an infant she was reported to have been healthy; her ailments began by inflammation of the nose after a blow at the age of 8.

Comments. I have not seen a case more abruptly-margined scars such as are present in this case. Yet there is not the slightest doubt that the patient has lupus vulgaris, for in some of the patches where disease is still present the apple-jelly condition is characteristic. As you have heard, the mother's history points to scrofula, and not to inherited syphilis. Yet in spite of this want of evidence we must diagnose syphilis. I never saw such destruction of parts, and such healing in connection with any other cause. I should suspect that in some way the syphilis was

puzzling as to diagnosis for a long time. The state of the patient's eyes, her absolute deafness, the extensive loss of bone which she has suffered, and the abruptly-margined conditions of many of her scars all point in the strongest possible manner to syphilis. With the necrosis of the alveolus her incisor teeth have been lost, so that we can obtain no help from them. The conditions which I have mentioned are no ordinary parts of strumous lupus, which, in the first place, never caused destruction of bone, and in the second never gives us sound,

acquired by accident in infancy, and that it was not inherited. Such cases do occasionally occur. You will see that this hypothesis will fit well with the facts, for the lupus might easily be a joint result of syphilitic taint and a tendency to tuberculosis. I have never seen so much destruction of the nose in a case of inherited syphilis, whilst it is not very uncommon in the acquired form of the disease. Everything, indeed, I think, points to this supposition. So far as I can see it is the only one which fits all the facts. The mother, however, tells us

that nothing went wrong with the vaccination, nor does she remember the occurrence of any illness until the nose was injured.

Lupus Erythematosus Treated by the Internal Use of Mercury.

I have brought this young woman here again in order to illustrate the beneficial effects of mercury upon the general health and upon the progress of lupus erythematosus. She has attended on several occasions before, and her case is a very typical one, and the results of treatment have been very interesting. Her lupus is now almost well, and has been so for the last six months. She took for eight months, quite regularly, three times a day, doses which contained a drachm of the solution of perchloride of mercury and a drachm of the tincture of bark. During the whole of this time she maintained her health, and gained in flesh and colour, whilst the lupus steadily receded. I had not seen her for six months, when she came to me a month ago, complaining of debility and breathlessness. This condition had come on when she was taking a tonic only without any mercury. I may say that she had suffered some years ago (before her lupus) from a definite attack of chlorotic anæmia. I am now giving her full doses of steel, and she is again improving. I have asked her to attend to-day, however, in order to prove that in a patient liable to anæmia a long course of mercury did not produce it, but had, on the contrary, an opposite effect. I believe there are many cases of delicacy, and especially those with strumous tendencies, in which the combination of mercury with tonics is very advantageous, both to the general health and to the local process. It improves the appetite and assists the secretions. The liver usually acts well, and there is no constipation of the bowels. Nor must we forget in any case in which we suspect the tubercle-bacillus to be present, that mercury is an universal parasiticide. In prescribing the solution of the perchloride, I believe that we are usually in the habit of giving very inefficient doses. I find doses of a drachm to two drachms very well borne, and I have many patients who are taking the latter without finding it necessary to observe any precautions as regards exposure to the weather. Usually, but not always, I combine it with an equal quantity of the tincture of bark. I am aware that it is possible that some decomposition takes place in this combination,

which renders the larger dose better borne, but I feel quite sure it is not such as to neutralize the mercurial effect.

THERAPEUTICAL NOTES AND FORMULÆ.

Lactophenin in Rheumatism and in Tabes Dorsalis.—Dr. G. v. Roth reports 28 cases of acute rheumatism treated with this substance : the doses given, were, in the height of the disease, 5 grams per diem ; as improvement set in the dose was gradually diminished to 3 grams per diem, or less. The best results were obtained in all cases.

The pain, local redness, and swelling disappeared within a very few days, usually in from 24 to 48 hours. The temperature was permanently reduced. In spite of the large doses, no unpleasant remote effects were observed.

It had also a good effect on some individual cases of chorea, and relieved the severe pains of tabes dorsalis very materially.

(*Wiener Klin. Woch.*)

Ureoidin in Uratic Arthritis.—Dr. H. Langstein has tried this substance in 50 cases of gout, including his own person, with very satisfactory results ; he thus summarises its advantages :—

1. It is easy and not unpleasant to take.

2. That even in large doses, 10 grams (about 2½ drachms) in the day, it has absolutely no unpleasant remote effects, except a slight aperient action rather beneficial than otherwise.

3. It has, after prolonged use, no irritating effect on stomach or heart.

4. It is pleasantly diuretic.

5. It is more certain than other means in discharging uric acid from the system.

6. It lessens the pain very markedly.

7. It is cheap.   (*Prager Med. Woch.*)

Diarrhœa in Infants.—For diarrhœa in infants from one to two years of age, the following is useful :—

R   Acid. carbolici   ...   ...   gr.ij
    Bismuth subnit.   ...   ...   ʒj
    Syrup acaciæ   ...   ...   ℥ss
    Aq. menth. pip.   ...   ad   ℥ij

M. ft. mist. : A half teaspoonful every 2 to 4 hours.

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